EXHIBIT A

REPORT: EVALUATION OF POTENTIAL PUBLIC HEALTH IMPACTS OF LEAD PRESENTED BY LEAD-CLAD TELECOMMUNICATIONS CABLES

BY

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TABLE OF ACRONYMS

Acronym	Full Name
AHHS	American Healthy Homes Survey
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BLL	Blood Lead Level
BLRV	Blood Lead Reference Values
CDC	U.S. Centers for Disease Control and Prevention
CPSC	U.S. Consumer Product Safety Commission
CTEH	Center for Toxicology and Environmental Health, LLC
CYA	cyanuric acid
DU	Decision Unit
FDA	U.S. Food and Drug Administration
EFH	Exposure Factor Handbook
GM	Geometric Mean
HUD	U.S. Department of Housing and Urban Development
IEUBK	Integrated Exposure Uptake Biokinetic Model for Lead in Children
ITRC	Interstate Technology & Regulatory Council
ISM	Incremental Sampling Methodology
MCL	Maximum Contaminant Levels
N	Total Number
NHANES	National Health and Nutrition Examination Survey
NYSDEC	New York State Department of Environmental Conservation

Acronym	Full Name
NYSDOH	New York State Department of Health
OSHA	U.S. Occupational Safety and Health Administration
POD	Point of Departure
RBA	Relative Bioavailability
RSL	Regional Screening Levels
UCL	Upper Confidence Limit
U.S.	United States
USDA	U.S. Department of Agriculture
US EPA	U.S. Environmental Protection Agency
USGS	U.S. Geological Survey
WSJ	*The Wall Street Journal*

TABLE OF SCIENTIFIC ABBREVIATIONS AND SYMBOLS

Abbreviation or Symbol	Full Name
cm	centimeter
dL	Deciliter
ft	Foot
in	Inch
kg	Kilogram
m	Meter
mg	Milligram
ml	Milliliter
L	Liter
oz	Ounce
Pb	Lead
ppm	parts per million
mg/kg	milligram per kilogram
µg	Microgram
µg/L	microgram per liter
µg/dL	microgram per deciliter
µg/m	microgram per meter
<	less than
>	greater than
~	approximately

TABLE OF RECENT SAMPLING REPORTS

Author:	Subject:	Location:	Date:
Eurofins Environmental Testing (Verizon contract)	Aerial Cables, Wappinger Falls	New York	2023a
Eurofins Environmental Testing (Verizon contract)	Aerial Cables, Coal Center and California boroughs	Pennsylvania	2023b
Eurofins Environmental Testing (Verizon contract)	Aerial Cables , West Orange	New Jersey	2023c
Exponent, Inc.	Exposed Surface Cables	Louisiana	July, 2023
Geosyntec Consultants	Exposed Surface Cables	Louisiana	August, 2023
Haley & Aldrich	Submerged Cables	California	October, 2021
NY State Department of Health	Aerial Cables	New York	July, 2023
Ramboll	Aerial Cables – Dearborn Height	Michigan	August, 2023a
Ramboll	Submerged Cables – Water	California	August, 2023b
Ramboll	Submerged Cables – Sediment	California	August, 2023c
Ramboll	Soil Lead – Bywater	Louisiana	October, 2023d
Ramboll	Aerial Cables – Trenton	Michigan	September, 2023e
Ramboll	Water and Sediment – Sugar Oaks	Louisiana	October, 2023f
Ramboll	Water and Sediment – Front St.	Louisiana	October, 2023g
Ramboll	Water and Sediment – Franklin	Louisiana	October, 2023h
Ramboll	Water and Sediment – Detroit	Louisiana	October, 2023i
Ramboll	Soil Lead – Donaldsonville	Michigan	September, 2023j
Ramboll	Aerial Cables – Dallas	Texas	June, 2024a
Ramboll	Aerial Cables – Denison	Texas	June, 2024b
US EPA	Aerial Cables	New Jersey	August, 2023

I. Professional Experience and Credentials in Toxicology.

My name is David Lee Eaton. I am currently Dean Emeritus of the University of Washington Graduate School, and Emeritus Professor of Environmental and Occupational Health Sciences, School of Public Health, at the University of Washington. I retired as a professor at the University of Washington in 2019 after 40 years, but have remained professionally active, including as a consultant in toxicology to government, academia, industry, and non-governmental organizations.

I have authored ~200 research papers, review articles, book chapters, books, and other publications focused largely on toxicology, including the "Principles of Toxicology" chapter for the principal textbook in the field: *Casarett and Doull's Toxicology: The Basic Science of Poisons* (Editions 3rd–9th, 1991–2018). I also served as co-author and editor of Volume 1, "General Principles," of the 15-volume series: *Comprehensive Toxicology* (3rd and 4th Editions, 2017, 2023). In addition, I recently authored a chapter entitled, "The Risk Assessment-Risk Management Paradigm," published in a major textbook in the field of public health: *Risk Assessment for Environmental Health*, 2nd Edition, 2022 (sponsored by The Association of Schools of Public Health). I have also held numerous leadership positions in the field of toxicology, including serving as President of the Society of Toxicology, and have received a number of honors and awards, such as my election to the National Academy of Medicine in 2011 and the 2024 Lifetime Achievement Award (Merit Award) from the Society of Toxicology.

I have spent much of my professional career helping students, professionals, regulators, and the public understand how chemicals in the environment, including lead, arsenic, pesticides, and other substances, might have adverse impacts on public health, depending on the specific circumstances of exposure and "dose." A full description of my publications, career activities, and honors and awards can be found in Appendix 1 of my report.

After publication of *The Wall Street Journal's* (WSJ) July 2023 articles discussing purported public health impacts from exposure to lead from lead-clad telecommunications (telecom) cables, I was retained by AT&T to evaluate potential health risks to the general public from the potential release of lead from the cables. More specifically, I was asked to review existing and new studies and data that evaluated the concentrations of lead in soil, water, and sediment, potentially emanating from lead-clad telecom cables, and to offer my professional opinions regarding any associated potential public health risks.

II. Executive Summary.

A. Approach.

As an expert in toxicology and risk assessment of chemical hazards, I reviewed both historical and recent validated data concerning the current state of knowledge about the public health impacts of lead, including available data on potential releases of lead from lead-clad telecom

cables. I have focused on childhood exposure to lead because it is widely recognized that children are the most sensitive to the adverse effects of lead, and for that reason, environmental health regulations of lead are typically based on estimates of childhood lead exposure. (Vig and Hu 2000; Needleman 2004; Bellinger and Bellinger 2006; ATSDR 2020; Flannery et al. 2020.) In the absence of any significant risk of lead exposure to children who are the most sensitive receptors, there would be no significant risk to adults.

B. Overall Opinion.

I provide a science- and data-driven assessment as to whether lead-clad telecom cables that have been in common use throughout the world for many decades present a significant risk to the public, especially children, who are most sensitive to environmental lead exposures. Based on my decades of experience, review of scientific literature, and recent validated data from numerous sites containing either aerial, buried, or submerged lead-clad communications cables, and utilizing conservative "risk averse" assumptions for each possible scenario, I conclude that the potential additional environmental exposures from lead-clad communications cables, if any, is negligible compared to background, and thus the additional risk, if any, is well below that from other common sources of lead in the environment deemed "acceptable" by regulatory agencies such as the United States Food and Drug Administration (FDA) and United States Environmental Protection Agency (US EPA).

C. Summary Opinions Based on Type of Lead-Clad Cable.

As detailed below, lead-clad cables lack the characteristics that would result in widespread dispersal of lead. They are typically located underground, underwater, or high overhead, and generally are not accessible to the public. Moreover, the studies and available data indicate that lead-clad cables contribute little to background levels of lead in surface soils or in waterways. Thus, they do not currently present—nor are they likely to in the future present—a significant source of lead exposure to the public.

In atypical circumstances where lead-clad cables or equipment are exposed to the surface due to weather or other circumstances and are accessible to the public, it may be prudent to cover the cables or take other action to limit the exposure risk. However, efforts to physically remove buried, submerged, or overhead cables are more likely to release lead into the environment than if the cables are left in place. I have seen numerous circumstances where efforts to reduce exposure to a substance in the guise of improving public health has led to actions that generate greater public health risks than would have occurred if the initial perceived risk was left unabated.

1) Buried Lead-Clad Cables.

Several studies have demonstrated that lead released from buried lead-clad telecom cables generally does not migrate in the soil more than a few inches from the cable. (Forsberg and Björkman 1994; Jaspers et al. 2001; EPRI 2004.) For example, Forsberg and Björkman (1994) found that, of the small amount of lead released to the soil immediately surrounding the buried

cable, 83% to 98% of lead remained within two inches of the cable. More recent studies have demonstrated that soil lead levels at a distance beyond several feet from lead-clad cables are no different than background levels of lead. (Jaspers et al. 2001; Nony 2018.) Furthermore, and as noted, cables are typically buried at least several feet below the surface, and any lead released from the cable cannot physically migrate upward toward the surface, as movement of lead in soil is largely due to water flowing downward because of gravity, taking small particles of soil with it.

I conclude, with a reasonable degree of scientific certainty, that buried lead-clad telecom cables do not present any significant public health risk because there is no viable exposure pathway. I do recognize the possibility of a buried cable becoming exposed to the surface, and I address those circumstances below.

2) Suspended (Aerial) Lead-Clad Cables.

Several recent studies have evaluated the presence of lead in soil beneath aerial lead-clad telecom cables. These have been reviewed in detail in Section VI.C. One study prepared in July 2023 by Ramboll[1] involved testing soils underneath aerial lead-clad cables located in Dearborn Heights, Michigan. (Ramboll 2023a, 2023e.) A second study prepared in July 2023 by the New York State Department of Health involved testing of soils underneath aerial lead-clad cables above a public park in Wappinger Falls, New York. (NY State Department of Health 2023.) This second location was discussed in the WSJ articles. Both Ramboll and the New York State Department of Health found no evidence that aerial lead-clad telecom cables significantly contributed to lead in soils. Another study was conducted in August 2023 by the US EPA in a neighborhood of older houses in West Orange, New Jersey. Although most of the soil samples collected from underneath both the lead-clad telecom cables and the "reference" areas had levels of lead that exceeded the new 200 parts per million (ppm) Regional Screening Level (RSL) for residential areas,[2] the concentrations were highly variable along the ~3,000 feet of sampling area, with adjacent samples ~15 feet apart varying by as much as 10-fold (from 130 ppm to 1,700 ppm). The variability suggests the impacts from alternate lead sources, such as lead paint debris from previous remodels of the 1930s-era homes and/or leaded gasoline from the past. Furthermore, the area was covered with grass, making lead in the top two inches of soil unavailable for direct skin contact. The US EPA's overall conclusion stated: "EPA's scientific review of the data and current conditions in the area indicate that there are no immediate threats to the health of people nearby."

[1] On September 1, 2023, Ramboll US Consulting transitioned to Ramboll Americas Engineering Solutions (RAES). The name "Ramboll" is used in this report to represent the company and its work products under either legal identity.

[2] See: https://semspub.epa.gov/work/HQ/100003435.pdf.

Additional studies in two of the locations noted above (New York, New Jersey) and in California and Coal Center Burroughs, PA evaluated lead concentrations underneath aerial lead-clad cables and are largely consistent with the previous studies discussed above (See Section VI.E for details).

More recently, two studies in Texas (Dallas and Denison) also demonstrated that aerial lead-clad cables contribute little, if any, additional lead to soils beneath them. Overall, the studies to date suggest that the impact of aerial lead-clad cables on surface soil lead levels beneath the cables appears to be modest and would not be expected to represent any significant threat to public health.

Thus, I conclude, with a reasonable degree of scientific certainty, that aerial lead-clad telecom cables present no significant public health risk because the additional exposure to lead from the cables, if any, would not add significantly to background exposure to lead from other sources.

3) Submerged Lead-Clad Cables.

Two studies in Lake Tahoe found low or no detectable levels of lead in the water adjacent to submerged lead-clad telecom cables. (Ramboll 2023b; Haley & Aldrich 2021.) It is important to note that submerged lead-clad cables have an outer protective covering that will impede the corrosion of the lead cladding. Another study involving collection of sediment samples near the same cables in Lake Tahoe found that lead concentrations in sediments near the cables were very low. (Ramboll 2023c.) The reports conclude that any lead detected in water or sediment near the cables was consistent with background levels.

An exposure assessment using the highest concentration measured adjacent to the cables (0.064 µg/L) and upper bound estimates of a person drinking water within inches of the cable during one hour of swimming (Dufour et al. 2017) would result in an estimated maximum level of exposure of 0.002 µg of lead, which is a small fraction (0.09%) of the FDA "Interim Reference Level" for daily intake of lead from food (2.2 µg/day for children and 8.8 µg/day for females of childbearing age. (Flannery et al. 2022.) Because of the physicochemical nature of lead used in the cables noted above, very little, if any, lead dissolves in water, and lead is not easily absorbed across the skin, either in soluble or particulate form. (EPRI 2004; ATSDR 2020.) Any dermal exposure that might occur, such as recreational users making contact with sediment or water with lead from the submerged cables, is not a viable exposure pathway because virtually no lead will be absorbed through the skin and into the blood.

In addition, because the vast majority of submerged cables are at depths inaccessible to swimmers, a person would be extremely unlikely to drink water immediately next to the submerged cables because water consumption during swimming typically occurs when swimming at the surface. Regardless, the level of exposure even under those worst-case conditions is extremely low. (Dufour et al. 2017.) Based on these data, I conclude, with a reasonable degree of scientific certainty, that submerged lead-clad telecom cables present no significant public health risk.

4) Surface-Exposed Buried Lead-Clad Cables

I understand that in unique circumstances, buried lead-clad telecom cables may become exposed to the surface by weather-related events or other causes. Although such exposure situations are not common, I address them in this report for the sake of completeness and because surface-exposed cables were a point of emphasis in the WSJ articles (e.g., Bayou Teche, Louisiana). I also understand that AT&T has covered the exposed cables and associated equipment in the Bayou Teche area.

Several studies have carefully evaluated the presence of lead in soil or sediment near surface-exposed lead-clad telecom cables. These studies have shown that lead can sometimes be found above background levels in soil near the cables. (Forsberg and Björkman 1994; Jaspers et al. 2001; EPRI 2004; Nony 2018; Exponent 2023; Geosyntec 2023; Ramboll 2023d.)

While data show that lead concentrations in soil or sediment samples immediately next to exposed cables at five sites in Bayou Teche, Louisiana were sometimes above background levels, samples collected 2 ft or more away from the cables were near or below US EPA's risk-based regional screening levels (RSL) for residential areas of 200 ppm, and well below the 800 ppm RSL for non-residential public areas. In a few instances, some surface soil samples within two feet of the exposed cable contained elevated lead levels in excess of the EPA RSL of 800 ppm for soils in public areas that are not residential. (Exponent 2023.) However, as discussed in Section V.G.(2), even using highly conservative assumptions (i.e., likely to overestimate exposure and risk), the soil lead data collected from five different surface-exposed cables/junction boxes demonstrate that worst-case exposures to lead from these sites would result in only negligible increases in blood lead levels.

Based on these considerations, I conclude, to a reasonable degree of scientific certainty, that surface-exposed lead-clad telecom cables and junction boxes, as represented by data from the five sites in Bayou Teche, Louisiana (Exponent 2023; Geosyntec 2023) and a sixth site in Bywater, Louisiana (Ramboll 2023d) will not add significantly to background exposures of children to lead and thus present no significant public health risk. However, the data from some sites showed lead from surface-exposed cables can elevate soil lead levels immediately surrounding the cables, and it would be prudent public health practice to identify and secure such sites to minimize public contact with affected soils in close proximity to the cables and boxes. (Exponent 2023.)

Overall, and as noted above, I conclude that lead-clad telecom cables do not present a significant public health risk. However, I note that my conclusions for three of the four scenarios above (excluding buried cables) are based on a relatively limited number of sites, and thus the generalizability of my conclusions would be strengthened by evaluation of additional sites.

III. Overview of Key Elements of a Public Health Risk Assessment from Chemicals.

A. Hazard Versus Risk.

In the fields of toxicology and chemical risk assessment, understanding the basic differences between "hazard" and "risk" is essential. Simply stated, "hazard" represents the scientific judgment, based on the weight of available scientific evidence, that a specific chemical or mixture of chemicals is capable of causing one or more specific adverse effects (i.e., a "toxic response") under some circumstances. Put another way, hazard is a qualitative judgment that "chemical A is capable of causing effect B," but it is dependent upon a number of assumptions that must be met before the hazard can be realized as an actual adverse effect. In contrast to hazard, the term "risk" refers to the probability or likelihood that a hazard will be realized (i.e., that the adverse effect will actually occur) under a defined set of circumstances, the most important of which is the dose and duration of exposure to the potentially hazardous substance.

Typically, a risk assessment includes both a characterization of the potential hazards that a chemical may pose (i.e., "Hazard Assessment") and a quantitative determination of the likely pathways or routes of exposure to the chemical (i.e., "Exposure Assessment"). From this information, a "Dose-Response Assessment" is performed to determine the amount of a chemical relative to the exposure frequency required for each potential type of hazard to occur (e.g., cancer, birth defects, neurotoxic effects, or any other of several possible adverse effects). The primary goal of the assessment is to utilize dose-response information to identify a dose (usually expressed in units of milligrams [mg] of chemical per kilogram [kg] of body weight per day or mg/kg/day) that would be associated with some small but measurable response (e.g., 10%) in the population exposed (either laboratory animals, in the case of basic "predictive toxicology" studies, or potentially in humans, usually using the tools of epidemiology to study occupationally exposed populations).

B. The Risk Assessment Paradigm.

The risk assessment paradigm for any hazardous substance includes both hazard identification (in a Hazard Assessment) and quantitative exposure assessment in addition to an estimation of the characteristics of dose-response. (Omenn and Eaton 2022.)

Typically, the dose-response step in the risk assessment process evaluates dose-response data from animal experiments (or, sometimes, human studies) to come up with an estimated daily dose that would cause an adverse response in a specific fraction of the population (e.g., 10%, 20%, 50%) if a population of humans were exposed under the same or similar conditions (e.g., dose, time/frequency of repeated exposures). This estimated dose is called a "Benchmark Dose" (BMD) or "Point of Departure" (POD) in classic risk assessments for chemical hazards.

The next step in a risk assessment is to carefully evaluate the assumptions and uncertainties that were implicit in the risk assessment—a process referred to as "Risk Characterization." As part of the Risk Characterization step, final prediction of the specific conditions necessary for an

adverse effect to occur are quantified, then "uncertainty" or "safety" factors are developed that reflect uncertainties in the data that were used to establish hazard and dose-response.

For example, risk characterization considers potentially important differences in how a species, such as the laboratory rat or mouse, responds to the chemical versus how humans respond. Because there are many biological similarities (and differences) in how laboratory animals and humans might respond to a chemical, each specific hazard (i.e., type of adverse response) might have different uncertainties, based on the biochemical and molecular processes that underlie the adverse response.

Once an estimate of a specific dose that would cause a specific level of response in the population (BMD or POD) is determined, the uncertainty factors are then applied to identify a dose level or dose rate that would likely be without adverse effects, even if exposure occurred daily throughout an individual's lifetime. Thus, using these types of risk assessment and risk characterization procedures, the FDA establishes "tolerance limits" for chemicals in foods, the US EPA establishes "Maximum Contaminant Levels" (MCLs) for drinking water contaminants, and the U.S. Department of Agriculture (USDA) and US EPA establish "tolerance levels" for pesticides used on food crops, in addition to other tolerance limits. In every instance, the Risk Characterization step includes the addition of uncertainty or safety factors that lower the dose, often by a factor of 10 to 100 or more, to ensure the screening level or guidance value is properly protective of human health. In other words, final "acceptable" levels of exposure (and thus risk) are almost always conservative–that is, they are likely to overestimate actual risk to ensure that unusually susceptible individuals in the population are not adversely affected. It is also important to recognize that such acceptable levels of exposure, when applied to an individual or a population, conservatively assume that that the individual(s) in the population are exposed each and every day to the substance at the stated "tolerance" or "acceptable" dose for most of their lifetime.

C. Risk Management.

Finally, once the Risk Characterization step is complete, the information is frequently utilized in regulatory settings to manage chemical exposure, and thus risk. Risk management is not entirely a scientific process, although it is heavily dependent upon the scientific information that goes into the Risk Assessment and Risk Characterization. Risk management also takes into account numerous other factors, including social, economic, and political considerations. (Omenn and Eaton 2022.)

IV. Overview of Potential Public Health Consequences of Excessive Lead Exposure.

A. Brief Background and Historical Perspectives.

The potential toxic effects of lead have been recognized since antiquity. (Montes-Santiago 2013.) But, as with every toxic substance, "the dose makes the poison." While the health

hazards of lead have been long recognized, based largely on relatively high levels of exposures in industries associated with the use of lead (e.g., lead smelting, use of lead as solder), the public health impacts of lead contamination of the environment were not really recognized until the 1960s. (Needleman 2004.) There is ample evidence that lead is a hazardous substance, with the most sensitive effect being potential impacts on brain development of children exposed to excessive amounts of lead *in utero* and/or during early childhood. (Vig and Hu 2000; Needleman 2004; Bellinger and Bellinger 2006; Crump et al. 2013; ATSDR 2020; Flannery et al. 2020.)

As discussed above, identifying something as a hazard provides little useful information beyond the type of adverse effect that might occur. More important is the consideration of "risk," which is the probability or likelihood that an adverse effect will occur under a given set of circumstances, the most important of which is "dose," or the amount of substance that is absorbed into the body.

Once a hazard is established for a specific toxic substance, a quantitative measure of the extent of exposure (how much and how often, which gives rise to dose) is required to make any reasoned judgment as to whether the presence of the hazardous substance presents any significant health risk. (Omenn and Eaton 2022; Brown et al. 2023.) From a public health perspective, a quantitative exposure assessment is critical to determine whether actions are required to intervene and lower the potential for excessive exposure. Merely identifying a possible hazard—in the absence of any assessment of exposure, dose, and risk—is largely meaningless and can lead to large misperceptions of risk among the general public. I note the WSJ articles did not include any scientifically valid quantitative exposure assessments or risk analysis. To the best of my knowledge, the WSJ has not released any information reflecting that its assertions are based on scientifically validated data or analysis. My analysis below is based on validated data and information in the scientific literature.

To provide context to my analysis, lead exposure to children has been recognized as a significant public health problem since the 1960s, with the vast majority of exposures emanating from three sources: (1) historical use of lead-based paint in residences and other buildings from the late 1800s to the 1970s and subsequent deterioration of the paint into lead-containing dust and particles; (2) extensive use of lead as a gasoline additive from the 1920s to the 1970s, leading to widespread contamination of lead in the environment caused by emissions from automobile tailpipes and other uses of leaded gasoline; and (3) local and regional distribution of airborne emission of lead-containing dusts and fumes from industrial operations associated with the mining, smelting, refining, and commercial use of lead. (Needleman 2004; Bellinger and Bellinger 2006; O'Connor et al. 2018; ATSDR 2020.) Additional periodic exposure to lead from children's toys, lead-based ceramics, lead used in indoor plumbing, lead from dietary intake and contaminated drinking water, and other household sources of lead also still occurs. (Bellinger and Bellinger 2006; O'Connor et al. 2018; ATSDR 2020.)

However, it is important to realize the incredible gains that have been made in reducing childhood exposure to lead over the last 50+ years. Figure 1 illustrates the remarkable decline in blood lead values in children since 1976–1980, based on the extensive "National Health and Nutrition Examination Survey" (NHANES), a nationwide representative sample of the U.S. population. (Egan et al. 2021.)

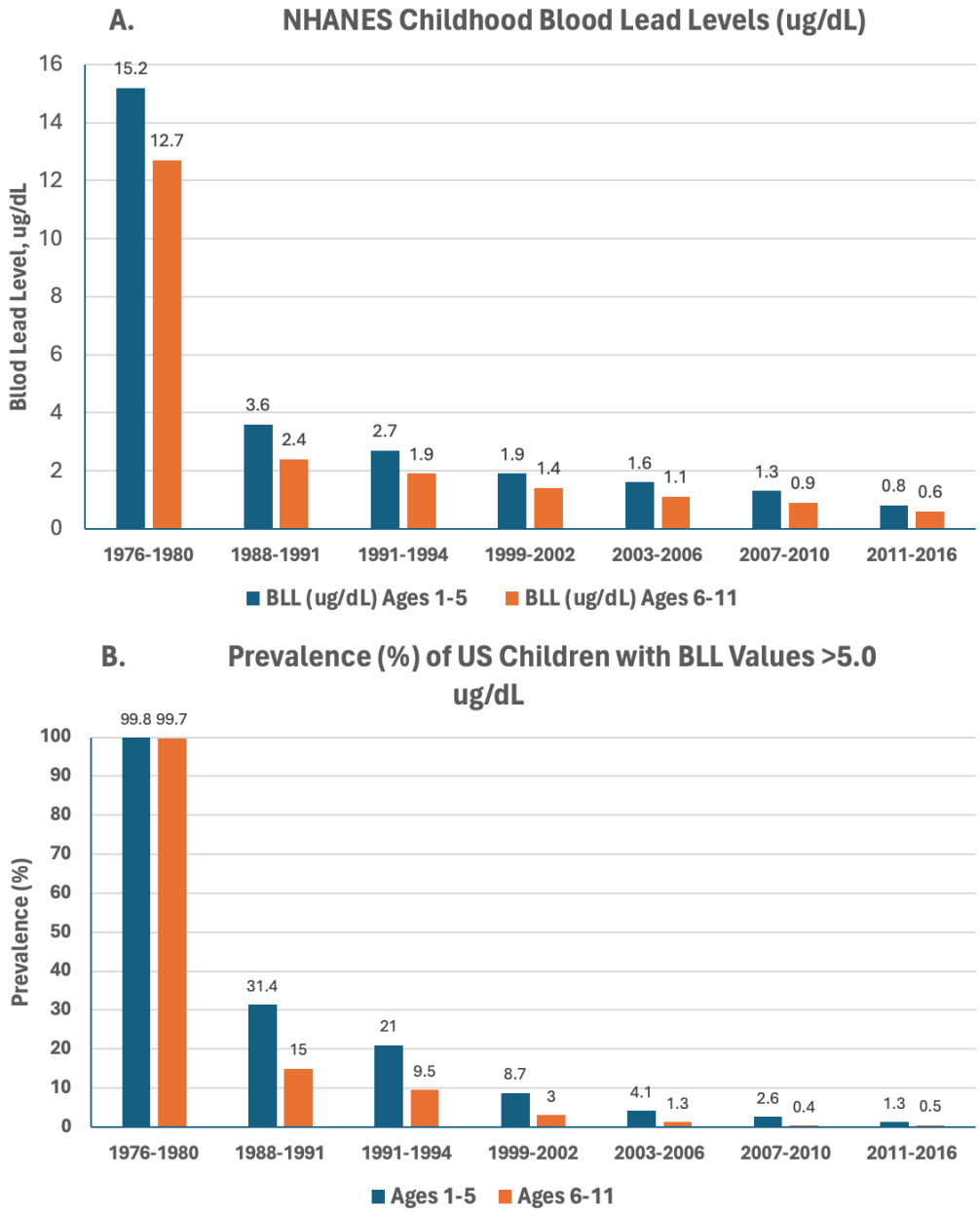


Figure 1. Historical decline in Blood Lead Levels (BLL) in US Children from 1976 to 2016. (From: Egan et al. 2021.)

This figure demonstrates how common (prevalence) it was in the 1970s and years prior for children to have blood lead levels (BLL) greater than 10 µg/dL. Virtually every young child in America prior to 1980 had a BLL greater than 5 µg/dL, and 90% had levels exceeding 10 µg/dL. The average BLL from 1976-1980 in children under the age of 5 in the U.S. was 15.2 ug/dL, an alarmingly high number from our perspective today. Over 99.8% of children ages 1–11 had BLL greater than 5 ug/dL. It is likely that such relatively high levels were commonplace in children for the previous several decades. With the recognition of the lead hazard in young children, efforts were implemented to eliminate lead from gasoline and paint, as well as less common, but nevertheless important, sources such as lead in children's toys and common household items, as well as contamination of drinking water from lead in older distribution systems. In the last NHANES survey (2011–2016) the prevalence of children with BLL above 5 µg/dL had declined to less than 1%, and those with BLL greater than 10 µg/dL, were less than 0.2%. The geometric mean BLL was 0.6-0.8 µg/dL in the latest sampling period. These values have likely decreased significantly in the 10 years since the most recent NHANES survey because older housing containing residual lead paint continues to decrease and other efforts to identify and reduce lead exposure in food and drinking water have also continued.

It is evident from these data that the significant public health problem of childhood lead exposure is largely a thing of the past, although it is completely appropriate to remain vigilant toward potential continued sources of exposure to lead. The challenge now is to identify—and take corrective action if necessary—significant sources of exposure that could, under real-world scenarios, actually increase a child's BLL to an extent that it would make a measurable difference in lifelong intellectual function. Although the current public health mantra is that there is no safe level of lead, this is largely derived by assumptions about a linear decrease in children's intelligence quotient (IQ) at doses below 5 µg/dL, based on evidence of a dose-related decrease in children's IQ at BLL above ~5 µg/dL. It should be noted that measuring statistically significant changes in IQ at BLL less than ~5 µg/dL is difficult, if not impossible, because the magnitude of effect, if any, is below the ability of IQ assessment tests to discriminate changes in IQ of less than 1 -2 IQ points in an individual, and thus in a population of individuals. For example, Figure 2 shows the results of a meta-analysis of all the major studies examining the relationship between IQ and childhood BLLs. (Crump et al., 2013; a reanalysis of data first presented by Lanphear et al. 2005.)

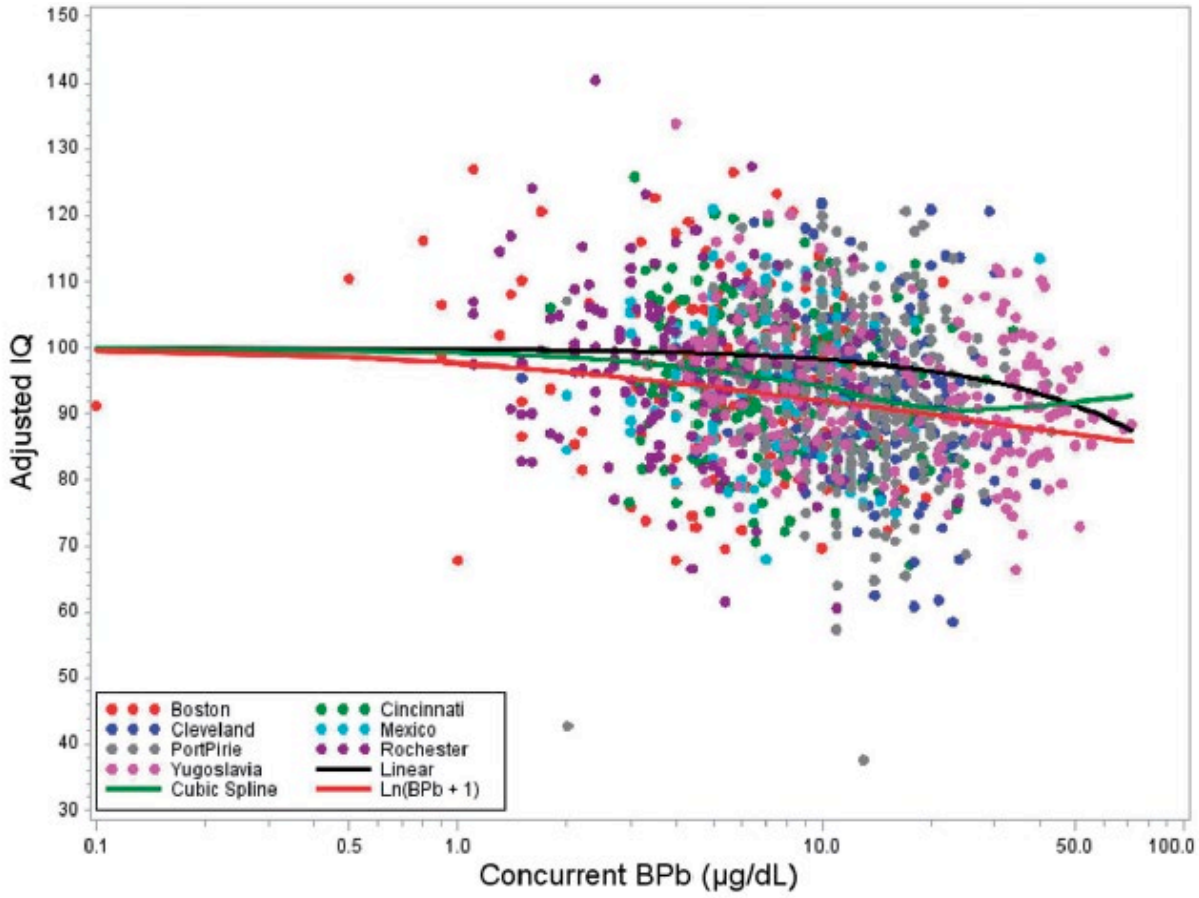


Figure 2. Plot of adjusted IQ by BPb with linear BPb, Ln (BPb.1), and 5-knot restricted cubic spline fits. (From: Crump et al. 2013.)

Note that the "X-axis" showing blood lead levels, in µg/dL, is on a log scale. It is evident that the loss of IQ with increasing BLL has a very small slope (e.g., small changes in IQ per unit BLL), and a large amount of variance across studies. Nevertheless, the data support the hypothesis that IQ continues to decline in a somewhat linear fashion at BLL below 10 µg/dL. But the projected magnitude of "effect" (decrease in IQ points) below the current BLL "reference level" of 3.5 µg/dL (which represents the 97.5th percentile of childhood BLLs based on the latest NHANES data) is less than 2 IQ points. It is widely recognized that the reproducibility of any intelligence assessment measurement is substantially greater than 2 IQ points. For example, a 2009 report from the Institute for Applied Psychometrics (McGrew 2009) estimated the concordance of different IQ assessments in the same individuals and reported a correlation coefficient of 0.706 (Figure 3).

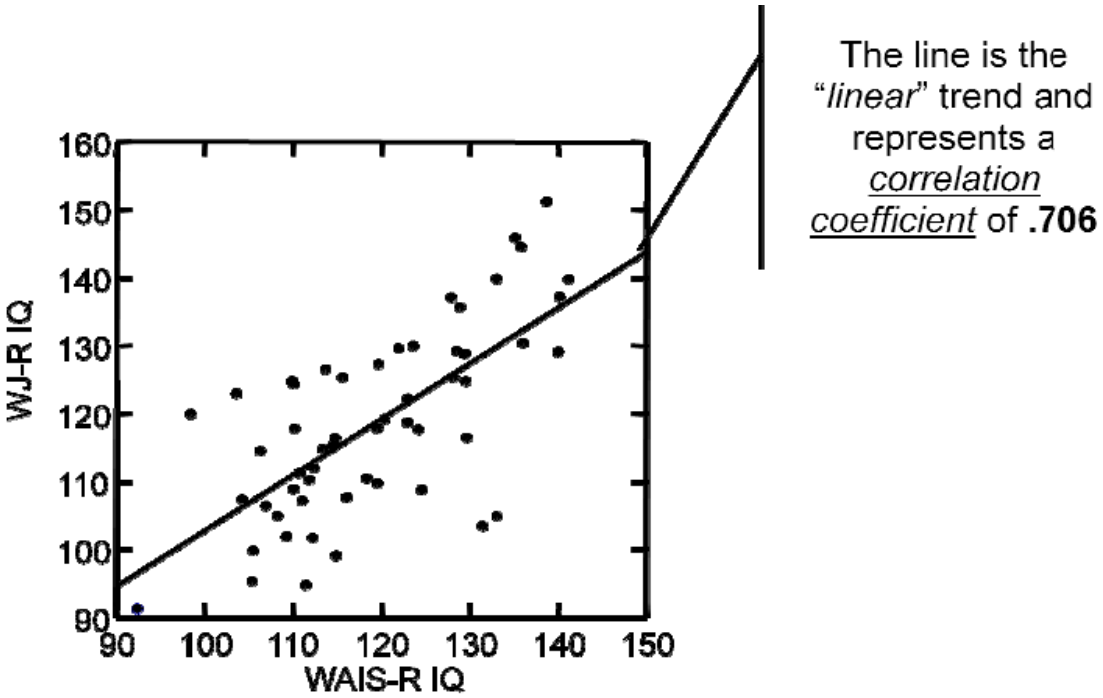


Figure 3. Scatterplot of two different Intelligence Quotient (IQ) tests (WAIS-R and WJ-R IQ) administered to the same 55 individuals. (From: McGrew 2009.)

While there is a strong statistical significance, between the two tests (r= 0.706), it is also evident that for any given individual the estimate of IQ could easily vary by 10 or more points based solely on which test was used. For about 20 of the 55 individuals in this study, the two tests gave results within 1–3 points, but for another 20, the difference was 10–30 points. Watkins and Canivez (2022) provide a detailed discussion of the challenges of reproducibly measuring IQ in children and the importance of proper interpretation of IQ measurements. McDermont et al. (2014) also evaluated the reproducibility of IQ tests in children, focusing on "evaluator error." They note in the abstract: *"This article reports on the application of multilevel linear modeling to examine the presence and extent of assessor bias in the administration of the Wechsler Intelligence Scale for Children—Fourth Edition (WISC–IV) for a sample of 2,783 children evaluated by 448 regional school psychologists for high-stakes special education classification purposes. It was found that nearly all WISC–IV scores conveyed significant and nontrivial amounts of variation that had nothing to do with children's actual individual differences and that the Full Scale IQ and Verbal Comprehension Index scores evidenced quite substantial assessor bias."* Duckworth et al. (2011) *"completed a meta-analysis of random-assignment experiments testing the effects of material incentives on intelligence-test performance on a collective 2,008 participants. Incentives increased IQ scores by an average of 0.64 SD, with larger effects for individuals with lower baseline IQ scores."* Note that, by design, the distribution of IQ scores is determined such that the average score is 100, and the standard deviation (SD) is set at 15. Thus, a variance of 0.64 SD is equivalent to a change in IQ score of 9.6 IQ units. The point here is that changes in estimated IQ of 1 or 2 points are not readily measurable and may be of limited, if any, functional significance to the individual (Watkins and Canivez 2022; McDermott et al. 2014; Duckworth et al. 2011). Nevertheless, it is prudent public

health policy to continue looking for sources of lead in the environment that could lead to exposures that significantly elevate blood lead. The question becomes how to focus on exposures that are biologically relevant versus those that are not likely to contribute to a biologically significant elevation in BLL, especially in the face of other known continual sources (soils contaminated from lead paint and/or leaded gasoline emissions, dietary sources, drinking water, lead-based ceramics or toys, food contamination, etc.).

B. Summary of Known Health Effects of Lead Exposure.

i. Adults.

Lead's toxic effects include headaches, constipation and stomach cramps (lead colic), muscle/joint pain, trouble sleeping, fatigue, irritability, and, at higher doses, peripheral nervous system damage (lead palsy). The vast majority of lead poisoning in adults comes from occupational sources, but some hobbies, such as shooting guns in indoor ranges, reloading ammunition, soldering and plumbing repairs, and use of lead-based glazes in ceramics, have on occasion caused lead poisoning in adults. (Vig and Hu 2000.) Although occupational exposures to lead are well-recognized and largely controlled, excessive exposure can still occur in some circumstances.

The hallmark biomarker for lead poisoning is the concentration of lead in blood ("blood lead" or "BLL"), typically expressed in units of micrograms (µg) of lead per deciliter (dL) of blood, or "µg/dL." Throughout most of the 20th century, BLLs in workers of less than 40–60 µg/dL were considered acceptable, based on the U.S. Occupational Safety and Health Administration's (OSHA) standard at the time. However, numerous studies have suggested that in the long term, lower levels of exposure to lead in adults may be associated with a variety of chronic diseases, including hypertension, cardiovascular disease, and psychiatric disorders, although it has been difficult to establish clear causal associations because of other confounding risk factors such as alcohol and tobacco use. (Hu et al. 2007.) Although BLLs are not particularly good measures of long-term exposure over many years because BLLs reduce over time, the values are reflective of cumulative exposures over recent months/years and continue to be the mainstay of exposure assessment for lead in both adults and children.

ii. Children.

Although lead poisoning from occupational exposures has been recognized for centuries, the susceptibility of children to lead, especially young children exposed in early life when the nervous system is developing, did not become evident until the 1960s. There are three principal reasons why children are more sensitive to the toxic effects of lead than adults:

1) When exposure occurs during early childhood, the brain is still rapidly developing and thus, it is more sensitive to environmental perturbations. This may also be in part because lead crosses the "blood-brain barrier" more effectively in children than adults.

2) Relative to adults, children absorb a higher fraction of ingested lead than adults because of physiological differences in the developing gastrointestinal, or "GI," tract.

3) Small children have more "hand-to-mouth" contact, and thus, have substantially higher levels of exposure to surface contaminants, including indoor house dust and soil (when adjusted for small body size).

C. Establishment of Blood Lead Reference Values (BLRV).

The CDC established BLRVs to identify children with higher levels of lead in their blood compared to most children. Due to the development of more sophisticated epidemiological and neurodevelopmental assessment tools and more stringent public health standards, what is considered the "acceptable risk" for a child's BLL has continued to decline over the past several decades, from a value of 10 µg/dL in 1970s to the current level of 3.5 µg/dL in 2021 (Egan et al. 2021) (Figure 4; See also Figure 1).

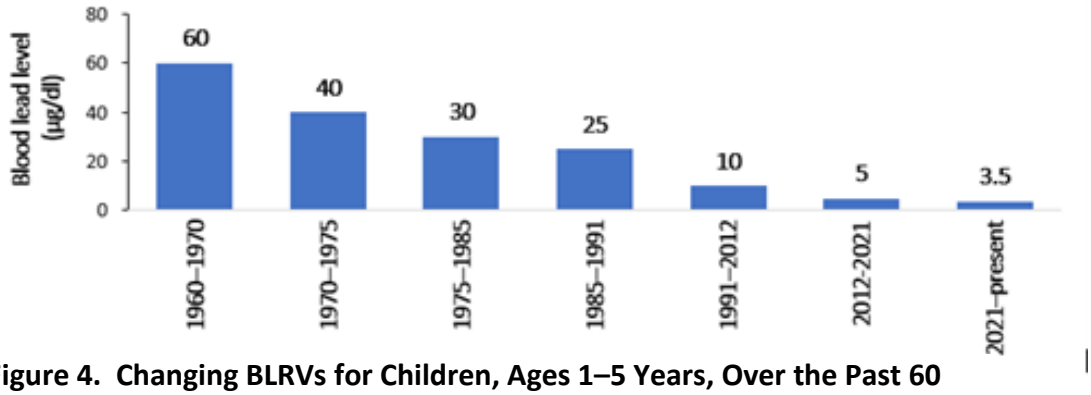


Figure 4. Changing BLRVs for Children, Ages 1–5 Years, Over the Past 60 Years. (From: Egan et al., 2021.)

From 2012 to 2021, the CDC's BLRV of 5 µg/dL was based on the 97.5th percentile distribution of the BLLs among U.S. children ages 1–5 years based on CDC's 2010 NHANES data. The current BLRV of 3.5 µg/dL is based on the 97.5th percentile of children's BLLs from the 2015–2016 and 2017–2018 NHANES cycles. Thus, children with a BLL at or above the BLRV represent those in the top 2.5% with the highest BLL (Figure 4).

The 10 µg/dL BLRV value established in 1991 for children exposed to lead was based on evidence of statistically significant adverse effects available at the time (based largely on small population shifts in IQ or learning ability in children above 10 µg/dL). However, when the value was lowered to 5 µg/dL in 2012, and then again to the current level of 3.5 µg/dL in 2021, these changes were based on statistical representations (the upper 97.5th percentile) of the distribution of BLLs in children 1–5 years of age in the U.S., and not specifically on demonstrated health effects. (Flannery et al. 2020.) Thus, while children with BLLs above 3.5 µg/dL do not necessarily suffer any adverse effects from lead, their BLL is simply in the upper 2.5% of blood lead distributions of children 1–5 years of age, and thus are assumed to be

potentially at risk, relative to 97.5% of the rest of the children. (Flannery et al. 2020.) These values are of significant use in helping parents identify possible sources of lead in their home environment but should not be interpreted as indicating that their child has lead poisoning.

A detailed review of more recent data supporting the associations between BLLs and adverse health effects is provided in the Agency for Toxic Substances and Disease Registry (ATSDR) Toxicology Profile for Lead (ATSDR 2020) and in a recent review from the FDA (Flannery et al. 2020).

D. Sources of Exposure to Lead that Contribute to Children's BLLs.

To determine whether a potentially new source of lead in the environment (i.e., lead-clad cables) could present a significant additional source of childhood exposure to lead, it is necessary to consider all known existing sources of lead. Because lead in the environment can exist in different forms that can greatly affect both the mobility and eventual absorption of lead into the body (thereby, contributing to elevated BLLs), it is also important to assess the physicochemical characteristics of the various sources of lead in the environment to determine, if exposure occurred, the relative contribution of a specific source to total BLLs in children.

Lead is a natural component of the earth's crust, with typical "geogenic" concentrations of lead in soil typically between ~10 and ~50 ppm.[3] However, there is a long history of known environmental (non-geogenic) sources of lead that are principally responsible for the elevated levels of lead in children. A brief summary follows.

1) Lead-Based Paint Used in Homes, Both Indoors and Outdoors.

Most homes constructed before the mid-1960s used lead-based paint, and thus older housing is often the most common source of lead exposure to children. It was only in 1978 that the U.S. Consumer Product Safety Commission (CPSC) banned paint intended for residential use with more than 0.06% lead by dry weight. Dried paint chips from decaying or peeling house surfaces in the past may contain lead at levels up to 10% lead by weight, although typical lead-based paint chips contain lead at slightly less than 1% (10,000 ppm). It has been suggested that lead-based paint was a more significant contributor to children's BLL than exposure to lead from automobile-based lead contamination of soil near roadways. (O'Connor et al. 2018.) The U.S. Department of Housing and Urban Development (HUD) found in its 2021 studies that lead in soil exceeding the US EPA RSL of 400 ppm in place at that time was present in 10.1% of homes nationwide. However, that number soars to 46.4% of all homes built prior to 1940, reflecting the presumed impact of lead paint. (HUD 2021.)

[3] US EPA document referencing USGS geogenic soil data across the US, available at: https://www.epa.gov/superfund/usgs-background-soil-lead-survey-state-data

2) Paint Used on Children's Toys.

ATSDR (2020) summarized several studies reporting lead in children's toys. Although toys and other home items (e.g., glazed pottery) may serve as an important source of lead exposure to young children, it is not a major contributor to the overall exposure to lead for the vast majority of children in the U.S.

3) Lead in Food Vessels (Pottery Glaze).

Lead has also been used for centuries as an amendment to pottery used as food vessels, usually in the paint or glaze used in the finish. This can be an important but usually rare source of significant childhood exposure to lead, as numerous studies have demonstrated that significant transfer of lead from pottery glazing to food items can occur.

4) Lead Exposure from Industrial Sources (Metal Ore Smelting, Refining, and Manufacturing).

Numerous studies have demonstrated that lead contamination of soil and indoor house dust from mining, smelting, and refining of ores containing lead can lead to widespread contamination of soils and indoor house dust in nearby communities, with lead levels as high as 13,400 mg/kg (or ppm) and 52,700 mg/kg, respectively. (Heusinkveld et al. 2021; von Lindern et al. 2016.) Thus, it is clear that environmental lead contamination from smelting and refining operations, as well as other lead manufacturing and use facilities, can be an important source of childhood lead exposure. (von Lindern et al. 2016.) However, much of this is now "legacy," as the majority of the ~1,000 lead-contaminated Superfund sites have been remediated.

5) Lead Exposure from Use of Leaded Gasoline (Tetraethyl Lead).

Another important and widespread source of environmental lead is a result of decades of use of "leaded gasoline." From the early part of the 20th century until 1976, tetraethyl lead was a common "anti-knock" additive for automobile gasoline.

In 1975, the US EPA issued regulations requiring a gradual phase-down in the amount of lead permitted in gasoline. However, the contributions of lead emitted from tailpipes to human lead exposure was not fully recognized until the late 1970s, when the second NHANES report revealed a precipitous decline in BLLs in all segments of the population, closely paralleling the decline in the amount of lead added to gasoline (Figure 5).

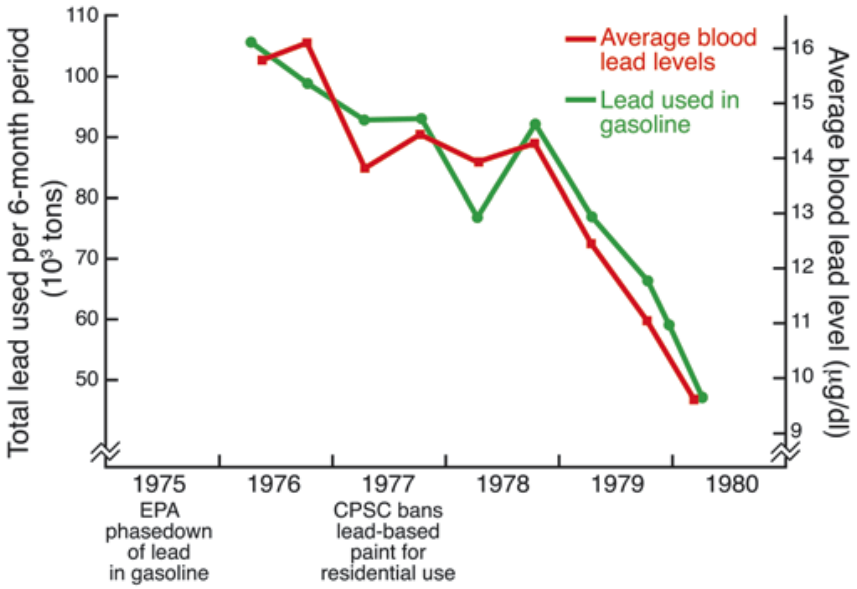


Figure 5. Declining BLLs in Children Following Bans on Lead in Both Paint and Gasoline.
(From: Bellinger and Bellinger 2006. See also Figure 1 for BLLs in children from 1996–2016.)

The combination of decreasing use of lead-based paint (and attention to careful remediation of homes containing lead-based paint) coupled with elimination of lead from gasoline has resulted in a remarkable decline in blood lead levels in children nationwide, as illustrated in Figures 1–5.

6) Mobility of Soil-Lead Derived from Automobile Exhaust.

Another important consideration when assessing the relative contribution of a particular source of lead to accessible pathways of exposure, such as indoor house dust or residential soil, is how easily it moves from the site or origin to possible receptor sites of potential exposure. Numerous studies have demonstrated that the concentration of soil-lead derived predominantly from combustion of leaded gasoline is highest near roadways, with rapidly declining levels as one moves away from the roadway.

7) Bioavailability of Lead in Soil.

An important factor to consider in risk assessment is the amount of bioavailable lead in the soil. "Bioavailability" is the fraction of the amount of ingested lead that is absorbed into the bloodstream. The fraction of total soil-lead that is bioavailable can vary substantially among different soils. The forms of lead in paint pigments are relatively soluble and thus have high relative bioavailability (>80%), whereas lead-contaminated dust from smelting and refining operations is generally much lower (<40%), with some forms as low as 8%. (Suedel et al. 2006.)

The bioavailability of lead in soil is determined by several factors, including the specific chemical form (species) present, particle size, and whether the lead material has been encapsulated or coated by other mineral phases. (Suedel et al. 2006.) Lead associated with larger soil particles is generally less bioavailable than when attached to smaller particles, and

encapsulation of lead mineral phases with clays or other materials can also reduce lead bioavailability.

8) Summary of Major Lead Sources Contributing to Children's BLLs.

In summary, it is evident that the major sources of lead in residential and urban environments are: (1) particulate remnants of lead-based paints; and (2) migration of fine particulate dust from roadways impacted by decades of combustion of leaded gasoline. Both sources contribute to indoor household dust and immediate exterior soils (e.g., yards and gardens). As shown in Figure 1 and Figure 5, above, studies have shown that these two sources are responsible for the vast majority of historical elevation of BLLs in children in the U.S., with obvious exceptions of children living in the vicinity of metal ore smelting, refining, or manufacturing facilities.

V. Assessing Potential Public Health Risks Specifically from Telecom Cables.

To estimate the potential threats (i.e., risks) to a child's health from lead exposure that might come from lead-clad telecom cables, one needs three types of information. First, the possible pathways of exposure must be identified (e.g., ingestion, inhalation). Second, the concentration of lead in the media of those pathways needs to be determined (e.g., soil, air, water). Third, one needs a reasonable estimate of the amount or volume of the media consumed (e.g., the amount of soil ingested, water consumed, or air inhaled). The dose then becomes the product of the concentration in each medium, multiplied by the amount/volume of the medium. The dose rate (how much is consumed over time) is also important. For example, lead intake in children is generally expressed in micrograms (µg) of lead per day, averaged over long periods of time (months to years). Ultimately, what is important is the impact of lead exposure to BLLs from the various media over weeks, months, or years. (Brown et al. 2023.) Once the information above is obtained, quantitative exposure estimates can be made, which are necessary to estimate potential public health risks. Estimating public health risks requires assumptions, many of which have scientific evidence to support them (e.g., how much soil a child ingests, how much water could be swallowed from swimming, and how much air a person breathes in an hour). However, some assumptions require reasoned judgment, often with limited data available. In such cases, common public health practice uses "conservative" or "risk-averse" assumptions to avoid underestimating possible risk. (Omenn and Eaton 2022; Brown et al. 2023.)

A. Basic Properties of Inorganic Lead Used in Cable Cladding.

The lead sheathing used in communications cables is present as a lead alloy, meaning the main chemical form of lead is a metallic lead. Metallic lead itself typically does not dissolve in water and is not "friable" (i.e., does not readily break down into a powder form). Over time, some of the exposed lead can become oxidized (corroded) to other chemical forms (e.g., lead carbonate) and form a passivating layer or protective film on the lead. This film acts as a retarding layer between the lead and the environment and will reduce the release rate of lead

into the environment. Over time, it is possible for small amounts of lead to be released from a lead-clad cable into the immediate environment through corrosion. (EPRI 2004.)

Another feature of lead is that dermal exposure is not a viable exposure pathway; virtually no lead is absorbed through the skin and into the blood. However, lead on skin can be ingested (e.g., hand-to-mouth) in circumstances, such as when an adult or child has lead on his or her hands through contact with affected soil and then later ingests that lead when using his or her hands to eat food, or in the case of small children, direct hand-to-mouth contact and/or mouthing of dust-contaminated toys.

I understand that lead-clad cables have been used for both telecom and electric transmission since the late 1800s. Among other functions, the lead sheathing serves as a moisture barrier to protect the inner copper wiring. In the 1960s, polyethylene and other coverings were introduced as alternatives to lead sheathing, but many legacy lead-clad telecom and electric cables remain in service throughout the world.

B. Possible Exposure Scenarios.

There are three possible sources of exposure to lead from lead-clad telecom cables:

1) Exposure to soils/sediments with lead from buried land cables that are directly buried and generally inaccessible to the surface

2) Exposure to lead in water or sediments from submerged cables in lakes or other water features.

3) Exposure to lead in soil coming from overhead suspended (aerial) cables.

4) Surface-exposed components of buried cables

In each of these possible pathways, it is essential to make reasonable, evidence-based analyses of the amount and frequency of exposure to lead that might occur under “real world” scenarios. Once a reasonable estimate of the possible amount and frequency of exposure to lead from a cable is determined, a comparison of that level of human exposure to other known sources of lead exposure (e.g., residential soil, house dust) can occur, and the potential impact of such respective exposures to BLLs in children can be estimated. From these two determinations, the potential magnitude of the incremental public health risk associated with the release of lead from lead-clad telecom cables can be reasonably assessed. In my evaluation of potential risks, I have used models and approaches adopted and currently widely used by both the US EPA and U.S. Centers for Disease Control and Prevention (CDC).

I understand that the majority of lead-clad telecom cables are buried, either directly in the ground or inside a conduit. Of those buried cables, most are inside a conduit (a barrier between the lead sheathing and the environment), which can prevent the release of lead into soil. I also understand that the cables underground in conduits generally are only accessible in secured buildings or manholes. For this portion of the telecom network, public exposure to

lead from lead-clad cables does not occur, and therefore I do not address cables in conduit further in this report.[4] I do address cables directly buried in the ground (not in conduit) because lead from the cables could possibly migrate into the soil.

C. General Assessment of Potential Pathways of Exposure to Lead from Telecom Cables.

1) Inhalation Exposure.

The lead in lead-clad telecom cables is in solid form and therefore does not readily release into the air. Therefore, any inhalation exposure to airborne particles of lead would be minimal and not provide any measurable impact on childhood BLLs (the final metric of concern in this assessment).

However, it is possible that land-based lead-clad cables, or soils directly beneath suspended or aerial lead-clad cables, could undergo degradation in a way that would release lead into surrounding soils, ultimately leading to airborne particles of dust with lead through wind dispersion of surface soil. Inhalation of lead-contaminated dust is generally considered to be a minor route of exposure to children, relative to hand-to-mouth contact leading to oral exposure, which will be discussed in the following section. (ATSDR 2020.) A historical exception to this could have been children living in the vicinity of lead mining, smelting, and refining operations that generated relatively high levels of airborne lead. (Brown et al. 2023; Heusinkveld et al. 2021.)

2) Drinking Water Exposure.

Because of the physicochemical characteristics of lead in lead-clad cables (water-insoluble lead complexes), it is highly unlikely that migration of lead from lead-clad cables, through soil, either directly or indirectly, into ground water occurs to any significant extent. However, consumption of small amounts of lake water from recreational activities such as wading or swimming could occur, albeit at very low volumes, and could theoretically serve as a pathway of exposure if lead from submerged lead-clad cables somehow migrated from the cable to the water's surface where swimmers might ingest lead-contaminated water. This is addressed below, with data from Lake Tahoe used to support the exposure analysis.

3) Dermal Absorption.

There is substantial evidence to demonstrate that dermal absorption of nearly all inorganic forms of lead is essentially zero and does not contribute significantly to childhood exposures to lead following contact with contaminated soil. (ATSDR 2000; US EPA 2004.)

[4] This report focuses on potential exposures to the general public. However, I observe that occupational exposures could occur to workers handling the cables in conduits, buildings, and manholes. I understand AT&T has policies in place to address occupational exposures and I do not address such exposures here.

4) Soil Exposure Via Hand-to-Mouth Contact.

As discussed at length by numerous reviews and government guidance documents (see, e.g., US EPA,[5] ATSDR,[6] CPSC[7]), soil contamination with lead is the most significant contributor to BLLs in children in the U.S. and throughout the world. There are a variety of mathematical models that have been used to predict what the BLL would be in a child who is exposed to lead-contaminated soil. There are also numerous variables for which either data or assumptions are used. The most widely used model is the US EPA's "Integrated Exposure Uptake Biokinetic Model for Lead in Children" (IEUBK). The purpose of the IEUBK model is to assist risk assessors (usually regional and state environmental protection employees and contractors who are involved with conducting or reviewing site-specific risk assessments at US EPA Superfund sites or equivalent state remediation sites) when determining whether soil remediation is necessary to protect the health of the public in the vicinity of the source of lead.

In 2021, Brown et al. (2023) did a detailed evaluation of BLL values in children residing in the Coeur d'Alene River basin in northern Idaho near the Bunker Hill Smelter, a major source of lead emissions for several decades. As described by Brown et al. (2023), the authors used the most recent version of the IEUBK model (v.2.0) that includes several sub-modules integrated into an overall model: *"An exposure module calculates daily intakes of Pb (µg/day) averaged over each year of age. Daily Pb intake is calculated based on the combined inputs for exposure concentrations or rates of intake of Pb in air, diet, indoor dust, soil, drinking water, or other user-defined sources of exposure. An uptake module calculates media-specific time-averaged rates of absorption of Pb to blood (µg/day). A biokinetics module simulates transfer of absorbed Pb between blood and other body tissues, and elimination of Pb from the body. The output of the biokinetics module is a GM BLL* [geometric mean blood lead level] *for each of the 84 months after birth. A variability module is used to calculate the probability of occurrence of a specified BLL over a user-specified age range in a population of similarly exposed children. This calculation is based on a lognormal distribution with the predicted GM BLL and a geometric standard deviation (GSD)."*

Several conclusions can be drawn from the Brown et al. (2023) evaluation of the IEUBK v.2.0 model:

1. The default assumption regarding drinking water concentration of lead has little overall impact, as it does not contribute significantly to blood lead value. The IEUBK v2.0 model default concentration for drinking water was 0.9 µg/L, but privately sourced drinking water concentrations in one of the study areas measured over a 12-year period averaged 2.3

[5] https://www.epa.gov/lead/lead-policy-and-guidance; https://www.epa.gov/sites/default/files/2020-10/documents/lead-in-soil-aug2020.pdf
[6] https://www.atsdr.cdc.gov/toxprofiles/tp13.pdf; https://www.atsdr.cdc.gov/csem/leadtoxicity/safety_standards.html
[7] https://www.cpsc.gov/Business--Manufacturing/Business-Education/Lead/Lead-in-Paint#:~:text=the%20children%27s%20product.-,Lead%20in%20Paint%20and%20Similar%20Surface%20Coatings,must%20also%20meet%20this%20requirement

µg/L, ranging from 0.9 to 8.8 µg/L (N = 55). Brown et al. (2023) concluded that the apparent underestimate of the average drinking water lead concentration of 1.4 µg/L (i.e., 2.3−0.9 µg/L) "*would have lowered the predicted GM BLL by <0.15 µg/dL"* and that *"[t]his small difference would not have appreciably affected model performance."* These conclusions provide strong rationale for concluding that lead contamination of source water (e.g., lake) with small, infrequent, ingestion amounts (e.g., 30–40 milliliters [ml], or ~two ounces [oz]) at low parts per billion (µg/L) concentrations would not have a measurable impact on BLL, even if it occurred on a daily basis over a period of years.

2. In a similar manner, inhalation of airborne lead generally has little impact on BLLs in children, unless there is a nearby industrial emission source of airborne lead. All children were assigned the IEUBK v2.0 model default value for lead in air of 0.1 µg/m^3. Annual averages in the industrial (mining) area for the period 1995–1998 reportedly ranged from 0.04 to 0.07 µg/m^3. Note that this is an area of the country substantially impacted by mining, smelting, and refining of lead, and thus had extraordinarily high levels of lead in soil. It is obvious that the relatively high levels of lead in soil have little impact on airborne levels of lead. Brown et al. (2023) concluded that "*[a]lthough these values are uncertain, this suggests the model may have overestimated exposures to air Pb and resulting in predicted GM BLL by <0.02 µg/dL (a negligible effect). Consequently, using IEUBK model default air values [0.1 µg/m^3] is unlikely to have introduced substantial error into the predictions of GM BLLs."*

3. Default values for dietary lead exposure in children are also included in the IEUBK v.2.0 model, with a default value being age-dependent and ranging from 2.7 to 6.0 µg/day (Table 1). The average (across all ages) default value in the IEUBK v.2.0 model (5.1 µg/day) was about 40% higher than the average (across all ages) of another study (Zartarian et al. 2017) of 3.06 µg/day used in the previous model. Brown et al. (2023) concludes that "*[a]t a soil Pb level of 200 mg/kg, the contribution of soil (and soil-derived house dust) to total Pb uptake is predicted to be ~53%, while the contribution of dietary Pb is ~42%."* Thus, assuming age-dependent daily soil ingestion rates (used as default values in IEUBK v.2.0 model) ranging from 94 to 52 mg/day (Table 1), ingestion of 70 mg/day (average across all ages) of soil containing 200 ppm (mg/kg, or µg/g) lead would yield a daily ingestion of ~14 µg/day of lead, of which ~30% is estimated in the model to be bioavailable, and thus the bioavailable lead from soil would be about 4.2 µg/day. It is not clear what the assumption used for bioavailability of dietary lead was, but it appears to have been ~75%.

4. The Brown et al. (2023) analysis of the IEUBK v.2.0 model provides the following, in terms of a generalized outcome of the model: "*The IEUBK v2.0 model predicts a GM BLL concentration of 2.3 µg/dL (P5 = 5%) when the soil Pb level is 200 mg/kg (and air, water and diet exposures are set to national default values).*"

Table 1. IEUBK Model Default and Alternative Values for Dietary Lead Intake and Soil Ingestion. (From: Brown et al. 2023, Supplemental Materials Table S1.)

Age (months)	Dietary Pb Intake (µg/day)		Soil Ingestion Rate (mg/day)	
	V2.0 default	Zartarian et al. (2017)	V2.0 default	EFH
6 to <12	2.66	0.70	86	70
12 to <24	5.03	2.58	94	90
24 to <36	5.21	3.44	67	60
36 to <48	5.38	3.54	63	60
48 to <60	5.64	3.57	67	60
60 to <72	6.04	3.85	52	60
72 to <84	5.95	3.80	55	60

EFH, values from US EPA Exposure Factor Handbook[8]

Thus, if air and drinking water contribute only a small amount (~5%) at "background" default concentrations, diet contributes ~42%, and total soil (indoor house dust plus outdoor soil) contributes about 53%, then a total soil concentration of 200 ppm lead would contribute to a blood lead value of about 1.2 µg/dL (0.53 x 2.3 µg/dL) (still below the 3.5 µg/dL BLRV), assuming that the average daily intake of soil is ~70 mg every day of the week, seven days a week for multiple years of childhood, and that *all* of that soil contained an average of 200 ppm of lead.

In reality, however, children will have highly varied daily exposure to outdoor soil (perhaps less variability to indoor house dust), the exposures will vary widely across days of the week and weeks of the year, and the likely concentration of lead in the soil will also be widely variable depending on source and site of contact with the soil and dust. From this one could then estimate that an increase in soil lead concentration of 100 ppm would theoretically increase BLL by ~0.3 µg/dL. Thus, if other sources of lead exposure (e.g., food, drinking water, air) contribute to a BLL value of ~0.6 µg/dL, and each 100 ppm of lead in a residential soil lead concentration increases BLL by ~0.3 µg/dL, a soil concentration of nearly 1,000 ppm lead would give rise to a

[8] https://www.epa.gov/expobox/about-exposure-factors-handbook#about

BLL of ~3.5 µg/dL. These assumptions become very important in assessing whether a particular exposure source, such as lead-clad cables, could reasonably present a significant contribution to the total exposure of a child to lead.

D. Potential Children's Exposure to Lead in Soil from Buried Telecom Cables.

The following section provides a description of validated available data on lead in soil potentially impacted by lead-clad telecom cables.

Lead-clad telecom cables buried in soil present a potential source of lead in soil. The important consideration from a public health perspective is the extent to which lead in buried cables leaches into soil and then migrates or is transferred upward to the ground surface to a site accessible to children.

As explained below, the vast majority of buried lead-clad cable will not present any potential risk to children, unless: (1) lead is released from the cable sheathing into the surrounding soil; and (2) the surrounding soil is disturbed in such a manner as to elevate the contaminated soil directly surrounding the buried cable to the ground surface. It should be noted that lead is also a natural component of the soil, with typical background levels with no anthropogenic contamination typically ranging between 10 and 50 mg/kg (USGS 2023), although surface soil samples in older residential areas and/or near roadways are typically higher. The US EPA has established 200 ppm lead in soil as representative of the "typical" concentration of lead in soil for use in the IEUBK model.

1) Soil Data from Studies on Buried Cable.

i. Historical data

Jaspers et al. (2001) provided an assessment of the ability of lead to migrate from buried lead-clad cables in Denmark. After identifying 13 different cable sites that had been in place for 35 years or more, the authors carefully exposed the buried cable and then measured the concentration of lead in soil at multiple locations adjacent to each cable.

In all but one site, levels of lead in soil were at or less than the background levels taken at each site 1.5 meters from the cable. In the case of the one site with levels above the background level, two samples measured 250 and 340 mg/kg of lead, but these were not immediately adjacent to the cable, and Jaspers et al. (2001) concluded: *"These are believed to be local concentrations and not caused by the cable."* The author's final conclusions regarding the subject were: "*The lead concentration in the soil surrounding thirteen lead-sheathed telecom cables with very long operational lifetimes, was determined. Five of the cables were buried in sandy soil, five in clay, and three in peaty soil . . . Eleven sites showed an almost constant lead concentration around the cable independent of the distance from the cable indicating no discernible influence of the cable on the lead concentration in the soil."*

An earlier investigation of the release of lead from lead-clad cables was reported by Forsberg and Björkman (1994). These investigators collected soil samples from below and above lead-clad telecom cables at seven different sites, with four different soil types of differing pH (from 4.8–8.0), in Sweden, and analyzed them for lead content. The cables had been installed 30 to 45 years prior to sample collection. Samples were collected at 5-cm (~2-inches) intervals from below, above, and diagonally below the cables at two locations for each site, approximately two meters apart. Not surprisingly, these investigators found that the concentration of lead was highest in the 0–5 cm interval directly surrounding the cable, with concentrations ranging between 100 and 2,700 mg/kg. This study demonstrated that the vast majority (83%–98%) of the lead in soil is retained within this 0–5 cm (~2 inch) interval in all soil samples. The highest lead concentrations were detected in the more acidic soils, and the lowest concentrations were detected in the more basic soils. An important finding in this study was that, even in low-pH soils, lead concentrations decreased dramatically to below 60 mg/kg in the 5–10 cm interval. The authors concluded: *"Therefore, any lead released from the cable was quickly immobilized within 5 cm from the surface of the cable."*

ii. **Recent data**

AT&T contract – Center for Toxicology & Environmental Health, Huntsville, TX: A study of the potential for lead to migrate into soil from buried lead-clad cables was conducted by the Center for Toxicology and Environmental Health (CTEH), LLC, an environmental consulting firm affiliated with the University of Arkansas for Medical Sciences BioVentures Program, retained by AT&T to prepare the study. (Nony 2018.) This study evaluated the concentration of lead immediately adjacent to a buried lead-clad telecom cable running beneath residential properties in a rural community northwest of Huntsville, Texas. The study involved collection of soil samples from a total of eight properties, of which the buried telecom cable transected six properties. On those six transected properties, CTEH identified the specific location of the lead-clad telecom cable, which was buried at a depth ranging from 3-4 feet below ground surface. Multiple soil samples (7–8 per property) were collected at the surface, six inches below the cable, and six inches above the cable on six of the eight properties. The results are summarized in Figure 6.

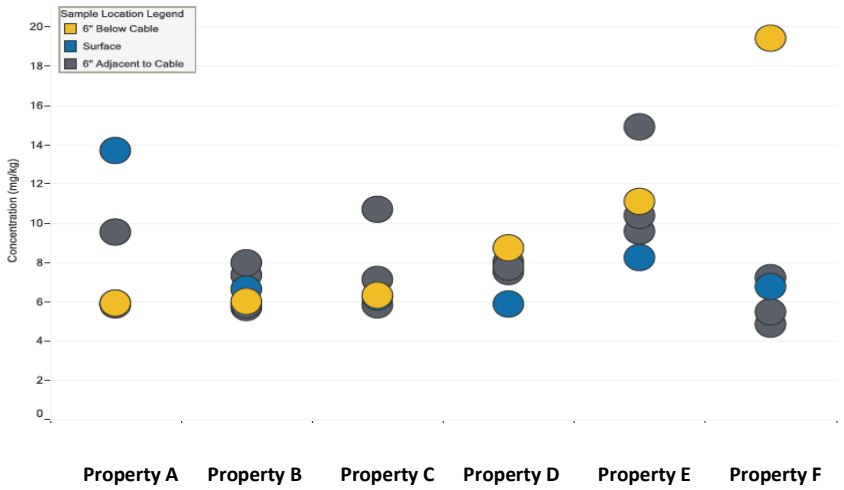


Figure 6. Soil Lead Concentrations Collected at the Surface (Blue), 6 Inches Below (Yellow), and 6 Inches Adjacent to (Gray) a Telecom Cable Buried 3-4 Feet Under 6 Residential Properties. (From: Nony 2018.)

The lead concentration in multiple background samples collected in the same general area ranged from 3.81 ppm to 45.2 ppm, consistent with general background soil lead levels in Texas and throughout the U.S. (Nony 2018.)

Two key points are evident from this data: (1) soils surrounding the cable, and at the surface immediately above the cable, had lead concentrations no different from multiple background soil sample levels (3.81–45.2 ppm); and (2) no evident lead concentration gradient is below the cable. These results further demonstrate that lead derived from lead-clad cables—if released to soil adjacent to the cables—does not leach from and contaminate soil beyond a small distance (<6 inches) from the lead-clad cable, and therefore is not a source for surface soil lead.

A study of lead in sediment immediately adjacent to submerged lead-clad telecom cables in Lake Tahoe, California was also conducted by Ramboll. (Ramboll 2023c.) Although the cables were submerged in water rather than buried in soil, the data on migration of lead from the surface of the cables into the sediment surrounding the cable provides additional information and data on the potential for lead to migrate out of a lead-clad cable. Ramboll collected 15 sediment samples that were placed five cm into the sediment at locations that were 15 cm, one meter, and two meters from the submerged cables (laying on top or slightly buried in the sediment, taking great care not to disturb the sediment during sampling). Specifically, samples were collected from three different locations on each of the two submerged cables in Lake Tahoe and then analyzed for both total and soluble lead. Samples were also collected at three different sites away from the cables as reference (background) locations, and sediment samples were also collected at four beaches not proximate to the cables.

Ramboll found the lead concentration in all samples to be very low (<10 ppm) and well within the range of typical sediment soil concentrations measured in the reference sites. Two of the samples collected near the "cut end" of the cables, which exposed the cable and lead sheath to the immediate environment, did have slightly higher lead levels compared to the samples collected along intact parts of the cables. However, the slightly higher values were still within the range of the reference samples and less than 10 ppm. Ramboll's study further demonstrates that lead in buried/submerged lead-clad cables has minimal mobility and does not significantly contaminate surrounding soil and sediment via migration away from the cable.

2) Potential Risk from Exposure to Lead in Soil from Direct-Buried Telecom Cables.

Given that the vast majority of buried lead-clad telecom cables are buried well below the surface, and that the migration of any lead released from a cable occurs slowly and is likely to

be in the downward direction because of infiltration of rainwater,[9] it is extraordinarily unlikely that lead released from buried lead-clad telecom cables could reach the surface at a level that would add significantly to existing surface soil lead present both naturally and from other anthropogenic sources.

As noted above, the existing data from multiple different studies demonstrate that migration of lead beyond a few feet from a buried cable is negligible in the absence of any physical disruption. These data demonstrate that buried cables do not significantly contribute to surface soil lead. Thus, buried lead-clad telecom cables do not pose any significant public health risk from lead or present a significant source of children's exposure to lead if the cables are left undisturbed.

E. Potential Children's Exposure to Lead in Soil from Aerial Telecom Cables.

Another extensive use of lead-clad telecom cables is for overhead transmission. Weathering of suspended cables could conceivably result in the contamination of soil with lead that sloughed off the cable or leached from the cable to the ground via rainwater. To assess the potential public health consequences of aerial lead-clad cables, an assessment of the concentration of lead in soil beneath these cables is required as part of an exposure and risk assessment.

1) Aerial Telecom Cable Exposure Assessment.

i. Historical Data.

No validated historical data was identified that assessed the concentration of lead in soils beneath suspended lead-clad cables.

ii. Recent Data.

a. Ramboll testing, Dearborn Heights, MI.

Ramboll also assessed the potential influence of certain aerial lead-clad cables by testing the concentrations of lead in soil beneath such a cable in Dearborn Heights, Michigan. (Ramboll 2023a.) Specifically, in July 2023, Ramboll conducted a field study in the area near the intersection of South Beech Daly Road and Norfolk Street in Dearborn Heights. Ramboll screened a section of the selected aerial cable for the presence of lead cladding to first confirm the cable was lead-clad. Ramboll then performed surface soil sampling directly beneath and alongside two lengths of the aerial cable containing lead and also collected background samples in a vacant lot down and across the street from the cable. As to the sampling, Ramboll's report provides: "*The sampling design followed the Interstate Technology & Regulatory Council (ITRC) Incremental Sampling Methodology guidance (ITRC 2020) which is relied upon by the U.S.*

[9] Multiple studies described previously sampled soil at different depths from the surface beneath aerial cables and/or away from buried lead-clad cables. In every instance, the data demonstrate that lead migration from the source (surface-contaminated soil or buried lead-clad cable) was (is?) limited to less than 3–4 ft downward from the source.

Environmental Protection Agency (US EPA). In accordance with the guidance, sampling grids, called Decision Units (DUs) were laid out to systematically collect soil subsamples which were composited together for lab analysis. The purpose was to produce representative soil samples directly beneath the cable and away from the cable . . . Triplicate composite soil samples, each made up of 30 subsamples, were collected to provide a robust understanding of potential soil lead concentrations." Samples were collected at/near the surface (0–2 inches) and just below the surface (2–6 inches). The results of the sampling were described as follows:

The first sampling area included DU-1, directly beneath the cable, and adjacent to DUs 2 and 3, moving away from the cable:

- DU-1 average: 52 ppm (0–2 inches) and 39 ppm (2–6 inches)
- DU-2 average: 33 ppm (0–2 inches) and 33 ppm (2–6 inches)
- DU-3 average: 44 ppm (0–2 inches) and 40 ppm (2–6 inches)

The second sampling area included DU-6, directly beneath the cable, and adjacent DUs 7 and 8, moving away from the cable:

- DU-6 average: 76 ppm (0–2 inches) and 47 ppm (2–6 inches)
- DU-7 average: 29 ppm (0–2 inches) and 21 ppm (2–6 inches)
- DU-8 average: 41 ppm (0–2 inches) and 29 ppm (2–6 inches)

The third sampling area was the reference area down and across the street from the cable, with two areas similar in size to those sampled beneath and near the cable (DU-4 and DU-5), and a smaller grassy area (DU-GS) between the street curb and the sidewalk:

- DU-4 average: 34 ppm (0–2 inches) and 31 ppm (2–6 inches)
- DU-5 average: 22 ppm (0–2 inches) and 26 ppm (2–6 inches)
- DU-GS average: 30 ppm (0–2 inches) and 49 ppm (2–6 inches)

In all instances, the lead concentration in surface soils underneath the cables was (average 46 ppm; range 29–76 ppm; N=6) slightly elevated relative to the concentration in surface soil in "control" areas a few blocks away from the overhead cables (average 29 ppm; range 22–34 ppm; N=3). Soil lead levels just below the surface (2–6 inches) averaged 35 ppm (range 21–47 ppm) underneath the cables, which were in the same range as subsurface soils in the control area (average 35 ppm; range 26–49 ppm). These results demonstrate that the overhead lead-clad cables have minimal measurable impact on soil lead levels. Although the average of six surface soil samples was somewhat greater than the control area, all samples were considerably less than urban surface soil lead levels in the vicinity of roadways (typically >100 ppm), and below the US EPA's new (2024) RSL of 200 ppm for lead in residential soils.

b. Ramboll Testing, Trenton, MI.

Ramboll also conducted a second study of soil lead underneath an aerial telecom cable in Trenton, MI. (Ramboll 2023e.) The design of the study was identical to that just described for the Dearborn Heights, MI study, so I will present the data in a similar manner:

The first sampling area included DU-1, directly beneath the cable, and DUs 2 and 3, were adjacent to but away from the area immediately beneath the cable:

- DU-1 average: 94 ppm (0–2 inches) and 60 ppm (2–6 inches)
- DU-2 average: 53 ppm (0–2 inches) and 24 ppm (2–6 inches)
- DU-3 average: 58 ppm (0–2 inches) and 31 ppm (2–6 inches)

The second sampling area included DU-4 and DU-5 which were at least 90 ft and 400 ft, respectively, from the closest lead-clad cable, and were considered "reference" sites.

- DU-4 average: 37 ppm (0–2 inches) and 32 ppm (2–6 inches)
- DU-5 average: 29 ppm (0–2 inches) and 26 ppm (2–6 inches)

Similar to what was found at the Dearborn Heights, MI site, the soil samples directly underneath the lead-clad aerial cable were modestly elevated compared to adjacent reference sites, but the magnitude of impact was marginal and of no public health significance.

c. New York Department of Health Testing, Wappingers Falls, NY.

Another study of soil lead levels beneath overhead suspended lead-clad cables was performed in an urban setting in New York. Following the WSJ report that identified "high levels" of lead in soil underneath aerial lead-clad telecom cables in a children's playground area in Wappingers Falls, New York, the New York State Department of Health, Division of Toxic Substance Assessment, conducted soil sampling analyses of this same area on July 13, 2023. (NY State Department of Health 2023.) Staff from multiple New York State agencies participated in sampling of soil for lead at the playground and adjacent areas in Wappingers Falls, New York. The investigators conducted X-ray fluorescence surface readings for lead and also collected 25 different soil samples for quantitative lead analysis.

The results and conclusions of this analysis of the potential for aerial lead-clad cables to contaminate the soil beneath them with lead was summarized by the New York Department of Health as follows: "*Twenty-five soil samples were taken at Temple Park, lead soil levels ranged from 50.6 to 410 parts per million (ppm). Of those, twelve soil samples were taken from within Temple Park play areas … that range from 58 to 283 ppm, which are all below the NYSDEC Restricted Residential Soil Cleanup Objective, applicable for active recreation, and HUD/EPA soil*

guidance value for children's play areas of 400 ppm[10] *. . . The G3 sample is from the soccer field and is considered a background sample due to its distance from any overhead cables. The six soil samples closest to the actual playscape . . . ranged from 58 to 82 ppm, again below 400 ppm. The ten soil samples directly under the cable . . . ranged from 51 to 410 ppm; these samples are along the roadway and outside of play areas so therefore more appropriately compared to the HUD/EPA general soil guidance value of 1200 ppm . . . The sample across Market Street . . . located near the lead sheathed cable that comes down to ground level had a value of 302 ppm. While this sample may be influenced by lead from the cable it is also close to the road (a common source of lead) and therefore it is not possible to clearly identify the source. Background samples along Market Street across from the park and down by the Highway Department ranged from 62 to 101 ppm. Overall, there was not a clear gradation of elevated lead underneath the cable and decreasing away from this line . . . but the data are limited."*

Based on that analysis, the New York State Department of Health made the following conclusion and recommendation: "*These results do not suggest a significant exposure potential or public health risk in children's play areas at Temple Park, therefore NYSDOH recommends that the park be reopened. The Village of Wappingers Falls should re-grass any spots of bare soil and continue to monitor the sufficiency of buffer materials (wood chips) and surface grass at the playground in the future."*

d. Verizon Wappinger Falls, NY.

Yet another study was done in this same Wappingers Falls, NY site, contracted by Verizon. (Eurofins Environmental Testing 2023a; Exhibit L to a Verizon report, Figure 7.[11]) Surface soil samples ranged from 65–380 ppm lead, with averages across the three different sampling sites below the cable of 90, 220, and 300 ppm, with an overall average of 203 ppm. The background levels at two sites ranged from 99–160 ppm lead, with averages for the two sites of 110 and 140 ppm, for an average of 125 ppm. Thus, the potential impact of lead on the soil beneath the cable was modest, with less than a two-fold increase from the two background samples, approximately equal to the new US EPA RSL for lead in soil in children's play areas of 200 ppm. (From Eurofins 2023a.)

[10] As noted previously, in January of 2024, the US EPA lowered the RSL for lead in residential soil from 400 to 200 ppm. https://semspub.epa.gov/work/HQ/100003435.pdf

[11] Eurofins Analytical Report, Confidential Tristate ISM project-NY, Job #240-188956-, 8/29/2023.

Sample #	Date	P-0-2[12]	S-0-2	T-0-2	Mean
WFNY -SU1	07/20/23	380	220	310	300
WFNY -SU3	07/20/23	110	65	83	90
WFNY -SU4	07/19/23	250	220	200	220
WFNY -BG1	07/19/23	110	99	110	110
WFNY -BG2	07/19/23	160	140	110	140
Sample #	**Date**	**4-8**	**10-14**	**22-26**	**34-38**
WFNY-SU1-SC1	07/20/23	740 (B)	100	54	43
WFNY -SU1-SC2	07/20/23	200	160	91 (B)	-
WFNY -SU1-SC3	07/20/23	110 (B)	130 (B)	19	34
WFNY -SU3-SC1	07/19/23	51	120	360	39
WFNY -SU3-SC2	07/19/23	98	300	62	18
WFNY -SU3-SC3	07/19/23	54	12	8.4	9.1
WFNY -SU4-SC1	07/18/23	150	86	360	120
WFNY -SU4-SC2	07/18/23	170	240	120	27
WFNY -SU4-SC3	07/18/23	370	130	240	51

Figure 7. Phase 2 ISM Vertical Soil Sample Locations – Temple Park, Wappingers Falls, New York – Market Street/Dutchess Terrace. (Figure and data table from: Eurofins 2023a.)

Sub-surface soil levels from core samples were again highly variable, with concentrations of lead ranging from less than 10 ppm to 740 ppm. Six samples exceeded the new EPA RSL of 200 ppm. However, it should be noted that these are not surface soil samples, so they do not represent any opportunity for exposure. Thus, the data from this site are consistent with the previous study by the New York State Department of Health and further support the conclusion of the New York State Department of Health that there are no significant public health risks from soil lead levels in this community.

The data from both the New York State Department of Health and the Verizon report, and the conclusions derived from them, are consistent with Ramboll's findings in Dearborn Heights and Trenton, Michigan that aerial lead-clad telecom cables do not contribute significantly to lead levels in soil. (Ramboll 2023a; Ramboll 2023e.)

[12] The designation of P-0-2, S-0-2, and T-0-2 in the upper part of the table simply represent triplicate sampling and analysis of the same area. Thus, the mean value is just the average of three samples taken at the same designated location. The designations in the lower pane of '4-8', 1'0-14', 2'2-26', and '34-38' represent the depth, in inches, from the surface.

e. US EPA testing, West Orange, NJ.

The US EPA also conducted a study of lead concentrations in soils beneath an aerial lead-clad cable passing through an older residential area in West Orange, New Jersey (Figure 8). (US EPA 2023c.) Most of the homes in this neighborhood were wood structures, built in the 1920s to 1930s. All of them were likely painted with lead-based paint over the years, and many were also highly likely to have been remodeled, repaired, or repainted, which may have involved sanding of painted surfaces prior to the 1970s, with little attention to containment of lead-borne particles. Further, the homes have been situated on trafficked city streets for most of the 20th century. Thus, it is not surprising that the lead content of surface soil, and even soil 6–12 inches below the surface, contained highly variable concentrations of lead in soil ranging from less than 20 ppm to over 1,000 ppm, with the median value of 340 ppm (median refers to the point where 50% of the samples are above or below). Out of 63 surface samples (0–2 in) collected in the US EPA study, three exceeded the US EPA's 800 ppm non-residential RSL. Most, if not all, of these were confined to very small areas, suggesting the impact of sources other than the aerial lead-clad cable. The US EPA also collected 17 surface (0–2 in) soil samples across the street from the aerial cable as control samples. These samples had a median surface soil lead concentration of 210 ppm.

Photographs of the area where the samples were taken demonstrate that the housing structures on the west side of the street were fewer and somewhat different than on the east side of the street, where the aerial cable samples were collected. Based on this difference, the aerial cable may have had some modest impact on the soil lead levels beneath them, although the variability in both the "control" (west side of the street) and the east side of the street where the aerial cabal was located was large (the standard deviation was 63% of the mean on the west side and 65% of the mean on the east side). Furthermore, the area was covered with grass, making lead in the top two inches of soil unavailable for direct skin contact.

Figure 8. US EPA soil testing in the West Orange, NJ aerial cable site. (From: US EPA 2023c.)

Thus, the impact, if any, was unlikely to increase the soil lead levels above the levels found in other urban reference areas. After completing its assessment, the US EPA concluded that its "scientific review of the data and current conditions in the area indicate that there are no immediate threats to the health of people nearby."

f. Verizon testing, West Orange, NJ.

Verizon also contracted with Eurofins Cleveland Environmental Testing (2023b) to conduct additional analyses of soil lead at the same West Orange, NJ aerial telecom cable site studied by US EPA (Figure 9).

Sample #	Date	P-0-2	S-0-2	T-0-2	Mean
WONJ-SU1	07/21/23	270	260	200	240
WONJ-SU2	07/21/23	300	190	290	260
WONJ-SU3	07/21/23	370	220	220	270
WONJ-BG1	07/24/23	230	250	270	250
WONJ-BG2	07/24/23	350	170	180	230
Sample #	Date	**4-8**	**10-14**	**22-26**	**34-38**
WONJ-SU1-SC1	07/12/23	490	280	77	8.5
WONJ-SU1-SC2	07/12/23	890	1200	45	16
WONJ-SU1-SC3	07/12/23	750	210	12	16
WONJ-SU2-SC1	07/12/23	240	530	35	75
WONJ-SU2-SC2	07/24/23	110	410	29	10
WONJ-SU2-SC3	07/21/23	570	140	27	42
WONJ-SU3-SC1	07/21/23	480	220	33	NR
WONJ-SU3-SC2	07/21/23	590	280	83	9.2
WONJ-SU3-SC3	07/24/23	470	120	37	6.4

Figure 9. Soil lead analysis – Verizon Phase 2 ISM and vertical soil core sample locations, West Orange, NJ. (Figure and data table from: Eurofins 2023b.) Note: P, S, T values in column labels represent sample replicates from 0–2, 4–8, 10–14, 22–26, 34–38 inch sample depths; values in ppm.

Surface soil samples underneath the cables ranged from 170–370 ppm, with average values of 240–270 ppm—no different from the background surface soil lead values collected at two sites not underneath the cable (230 ppm, 250 ppm). Subsurface core samples showed highly variable lead concentrations in the first 14 inches of soil (ranges from 110–1,200 ppm), whereas deeper soil levels were typical of natural lead levels in soils (~10–80 ppm) not impacted by

anthropogenic activities (e.g., lead paint, leaded gasoline, industrial activities, etc.). Overall, the data from the West Orange, NJ site are consistent with the previous US EPA study of the same site, demonstrating that there was no significant impact of the aerial telecom cables on the levels of lead at the surface, where potential exposure to soil lead could occur.

g. US EPA/TetraTech/Verizon Coal Center and California Boroughs, PA sampling.

The US EPA contracted with TetraTech, Inc. to conduct soil sampling in two adjacent areas in Pennsylvania, Coal Center Borough and California Borough, PA, underneath an aerial lead-clad cable (Figure 10).

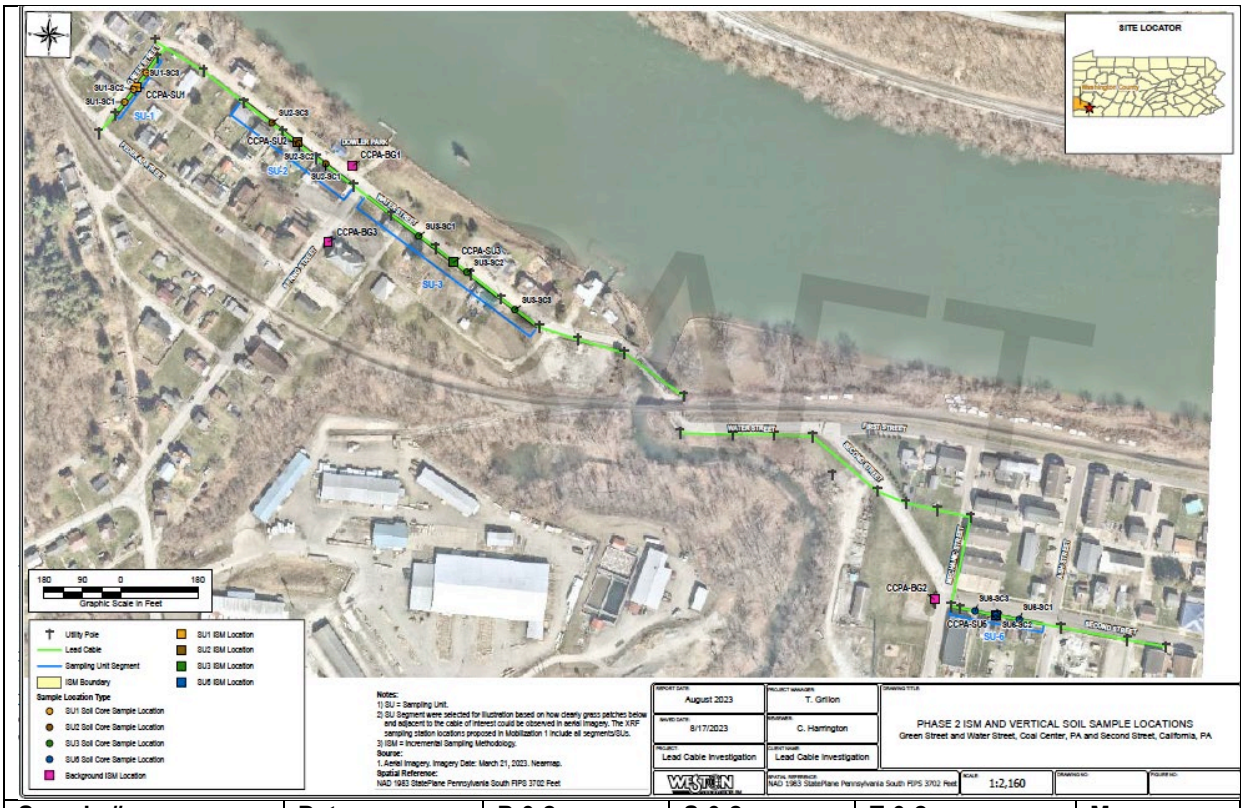
For the California Borough, 20 surface soil samples were collected beneath the aerial cable, with values ranging from 48.4 to 1,100 ppm. The average concentration of the 20 samples below the cable was 267 ppm (223 ppm if the one outlier at 1,100 ppm is excluded). Fifty percent of the samples beneath the cable were below 200 ppm, with 40% between 200 and 400 ppm. The lead concentration in the 5 reference ("background") sites ranged from 44–303 ppm, with an average value of 139 ppm. These results suggest that there was some anthropogenic contribution of lead to the soil beneath the cables, with modest impact on overall soil concentrations, approximately doubling the soil concentration relative to background samples that were collected in adjacent areas not underneath the cable.

California Borough		Coal Center Borough	
Sample ID	Validated Lab Results (mg/kg) [4] Method SW846 6010D	Sample ID	Validated Lab Results (mg/kg) [4] Method SW846 6010D
CCC-SS-01	195	CCC-SS-21	249
CCC-SS-02	408	CCC-SS-22	432
CCC-SS-03	187	CCC-SS-23	547
CCC-SS-04	185	CCC-SS-24	139
CCC-SS-05	157	CCC-SS-25	230
CCC-SS-06	180	CCC-SS-26	274
CCC-SS-07	256	CCC-SS-27	79.5
CCC-SS-08	198	CCC-SS-28	262
CCC-SS-09	48.4	CCC-SS-29	276
CCC-SS-10	135	CCC-SS-30	1250
CCC-SS-11	381	CCC-SS-31	1440
CCC-SS-12	1100	CCC-BKG-06	84.1
CCC-SS-13	273	CCC-BKG-07	831
CCC-SS-14	317	CCC-BKG-08	71.5
CCC-SS-15	222	CCC-BKG-09	192
CCC-SS-16	90.8		
CCC-SS-17	371		
CCC-SS-18	262		
CCC-SS-19	88.9		
CCC-SS-20	286		
CCC-BKG-01	141		
CCC-BKG-02	156		
CCC-BKG-03	50.6		
CCC-BKG-04	44		
CCC-BKG-05	303		

Figure 10. Lead Concentrations Found in Soil Samples at California and Coal Center Burroughs, PA. (From: TetraTech, Inc 2024, under contract to US EPA.)

In the Coal Center Borough, eleven surface soil samples were collected underneath the suspended cable, with validated values ranging from 79.5 to 1,440 ppm. The four background samples ranged from 71.5 to 831 ppm, with an average value of 295 ppm. Given that two adjacent samples had relatively high levels of lead (>1,000 ppm) but immediately adjacent were two samples with levels typical of the rest of the samples and the average of the adjacent background samples, there likely was some specific anthropogenic source of lead that contributed to very localized contamination. Since one of the adjacent "background" samples also contained relatively high levels (831 ppm), there was likely another anthropogenic source besides the lead-clad cables that contributed to the three "high" samples.

Verizon contracted with Eurofins Cleveland Environmental Testing (2023c; Exhibit N to Verizon report) to conduct soil sampling underneath an aerially suspended telecom cable located at Green Street and Water Street in Coal Center, PA and Second Street, California, PA (Figure 11). This appears to be identical to the location discussed above for US EPA's sampling (contracted to TetraTech).



Sample #	Date	P-0-2	S-0-2	T-0-2	Mean
CCPA -SU1	07/26/23	350	390	310	350
CCPA -SU2	07/26/23	610	340	380	440
CCPA -SU3	07/26/23	330	310	340	330
CCPA-SU6	07/26/23	300	530	480	440
CCPA -BG1	07/26/23	49	45	46	45
CCPA -BG2	07/26/23	170	160	150	160
CCPA -BG3	07/26/23	490	500	480	490

Figure 11. Verizon sampling in Coal Center and California districts, PA. (Figure and data table from: Eurofins Environmental Testing 2023b.)

At the Coal Center, PA site, triplicate soil samples were collected at four different locations beneath the aerial suspended telecom cable. The values of the 12 samples ranged from 300–610 ppm lead, with averages at four surface soil sites ranging between 330–440 ppm lead (Table 2). It is of interest to note that there was a large variability in the background level of lead at three different sites not beneath the areal cable. One site averaged 45 ppm, indicating little anthropogenic impact of lead on soil; one averaged 160 ppm, indicative of typical urban background levels of lead; and one site averaged 490 ppm, indicative of some anthropogenic impacts of lead unrelated to the presence of the lead-clad aerial cable. Ten of the 12 sites underneath the aerial cable were less than the highest background sample and the average values in all four of the surface soil samples were less than the average of one of the background sites. Anthropogenic input from sources other than lead cables was highly variable, indicative of significant of significant anthropogenic input. Thus, it is difficult to determine whether the aerial cables contributed significantly to all anthropogenic sources of lead in the area.

Subsurface soil levels at this site were, again, highly variable, with samples ranging from 45 to 950 ppm in the top 14 inches. Overall, the results of these two studies of the same area were consistent with the presence of lead in soil in urban areas typically ranging from 150-400 ppm, with a few notable "outliers" seen in both studies. Such levels are typical of most urban soil lead levels in neighborhoods with older housing (pre-1975) and residential streets.

As with other studies, there was a large degree of variability in soil lead levels across the various sampling sites, with adjacent values sometimes differing by five-fold or more, indicative of highly variable anthropogenic sources of lead to surface soil in this urban environment. Given that background soil levels from the various sites tended to bracket the vast majority, but not all, of soil lead values from samples collected immediately underneath the aerial cable, it is difficult to conclude that the occasionally "elevated" soil lead level was elevated solely because of the presence of the aerial cable. Other common anthropogenic sources, such as lead from leaded gasoline or dust emanating from lead-based house paint, could have contributed.

h. Caravanos et al. XRF analysis of soils at NY, NY, and PA sites previously evaluated by others.

In March of 2024, another report was published by Caravanos et al. (2024), titled "Measurement of Soil Lead Levels Adjacent to Lead-Sheathed Communications Cables." The results of the sampling data, using XRF rather than laboratory analysis, from this study of three different aerial lead-clad cables are shown in Table 2.

Table 2. Lead in soil levels within select New Jersey (NJ), New York (NY), Pennsylvania (PA) and Louisiana (LA) communities with aerial and submarine lead-sheathed communications cables (in mg/kg or ppm, estimated by X-ray Fluorescence [XRF] analysis). (From: Caravanos et al. 2024.)

Location	Type	Total Number of readings	No. readings above background	Mean ± SD	Readings above 200 ppm[a]	% Readings above 200 ppm[a]	Maximum	EPA background (95% UCL)[b]	$^{206}Pb/^{204}Pb$ background[c]	$^{206}Pb/^{204}Pb$ LSCC[c]	$^{206}Pb/^{204}Pb$ sediment/soil[c]	Different from background
Site 1 NJ	Below aerial LSCC	315	289	403 ± 337	209	72.3 %	3,055	71.3	18.988	19.666, 20.686	18.913, 19.215	Inconclusive
Site 2 NY	Below aerial LSCC	209	200	268 ± 254	88	44.0%	1,634	41.0	18.748	19.169	17.516	Inconclusive
Site 6 PA	Below aerial LSCC	52	52	602 ± 660	41	78.8 %	4,087	61.4	18.881, 19.059	18.165, 18.160, 18.515	18.182, 18.288, 18.763	Yes
Site 3 LA-D	Submarine LSCC	47	47	697 ± 866	31	66.0%	4,151	21.2	NA	NA	NA	NA
Site 4 LA-H	Submarine LSCC	61	61	159 ± 280	11	18.3%	2,070	21.2	NA	NA	NA	NA
Site 5 LA-N	Submarine LSCC	53	53	366 ± 467	15	28.3%	5,415	21.2	19.151	20.437	20.592	Yes

Note: EPA, US Environmental Protection Agency; LSCC, Lead-sheathed communications cable; NA, not applicable, not measured; NIST, National Institute of Standards and Technology; Pb, lead; ppm, parts per million; SD, standard deviation; 95% UCL, 95% upper confidence limit.
[a]On January 17th, 2024, the US EPA Lead in Soil guideline (screening level) was lowered from 400 to 200 ppm.
[b]US EPA background (95% UCL) data from "Background Soil Lead Survey State Data."[7]
[c]Pb corrected for instrumental mass fractionation to NIST 997 205Tl/203Tl = 2.38714 by exponential mass fractionation law and to NIST 981 = 16.9356, 15.4891, and 36.7006 for 206Pb/204, 207Pb/204Pb, and 208Pb/204Pb, respectively, by sample-standard bracketing. External reproducibility for Pb isotope analyses = ± 250 ppm.

No details on specific sampling locations are provided in the article, although it appears that these three sites in New Jersey, New York, and Pennsylvania are the same sites as discussed above. Because the paper provides no analytical validation of the XRF data and also provides no comparison to local "background" samples collected in areas away from the cable but rather relies on US EPA geological background soil lead levels for comparison, it is difficult to draw any significant conclusions from this paper regarding the impact of aerial lead-clad cables on the soil immediately beneath them.

i. Aerial Cable Lead Study, Dallas, TX.

In June of 2024, Ramboll conducted additional soil sampling and analysis beneath an aerial lead-clad cable immediately adjacent to a four-lane residential street in Dallas, TX. (Ramboll, 2024a.) Samples were collected from the surface soil (0–2 in) and subsurface soil (2–6 in) in three DUs: DU1 was immediately adjacent to the street and underneath the suspended cable, DU2 was adjacent to DU1 but away from the street, and DU3 was adjacent to DU2 and further from the street. Each DU was 12 ft wide and 117 ft long, except for DU1, which was 6 ft wide because the adjacent roadway surface underneath the cable prevented soil sampling (Figure 12).

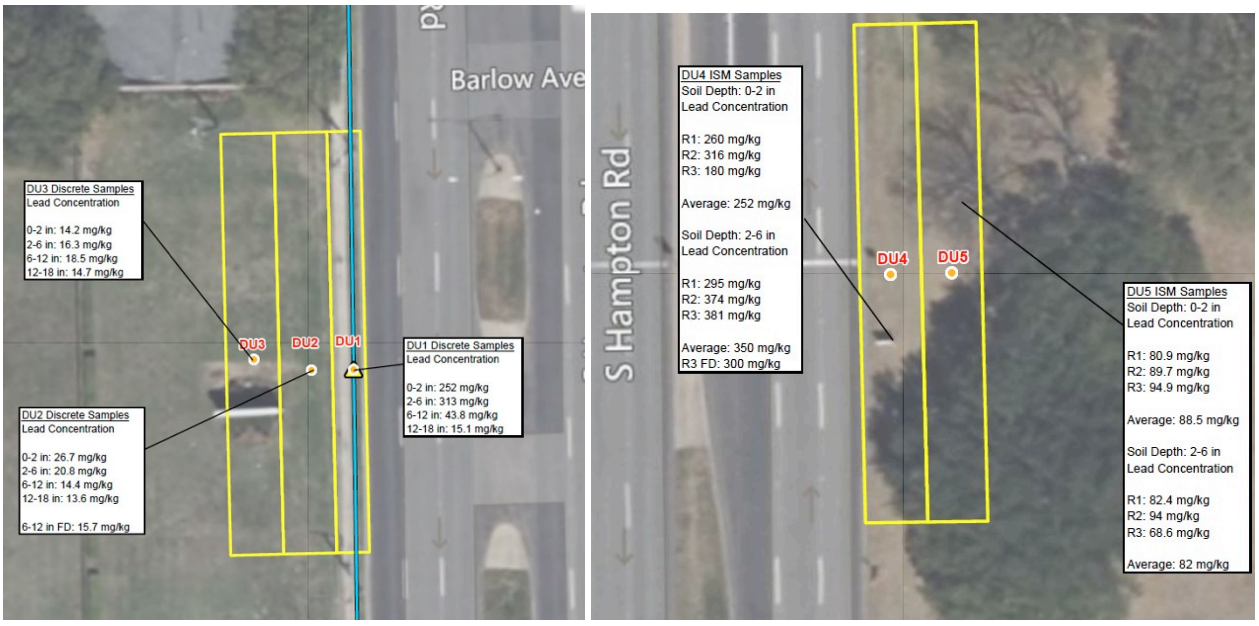


Figure 12. Soil sampling beneath Aerial Cable (left, DU1, DU2, DU3) and Control site (DU4, DU5) in Dallas, TX. (From: Ramboll 2024a.)

Two types of soil samples were collected in each DU: 1) Incremental Sampling Methodology (ISM) which is composed of 30 "cells" from which surface (0–2 inch) samples were collected, and 2) Discrete Soil Samples, which were core samples divided into depths of 0–2 and 2–6, and then an additional four samples collected at 6-inch depth intervals to a final depth of 36 inches. The results of the discrete soil samples at various depths are shown in Figure 12 for each of the five DUs. ISM results provided very similar numbers to the 0–2 inch discrete samples. For example, in DU1 next to the roadway and underneath the cable, the discrete sample measured 252 ppm, whereas the three ISM composite samples for DU1 averaged 235 ppm with a range of 195–284 ppm. DU2 values, which were both away from the street and not underneath the cable, had a discrete surface (0–2 inch) sample value of 26.7 ppm, whereas the ISM lead values were somewhat higher, with an average of 66.7 ppm (range: 45.4–90.5 ppm). DU3 values, further away from both the cable and the street, were lower than DU2, with a discrete value of 14.2 ppm, typical of background soil (ISM values for DU3 ranged from 31.2–39.8 ppm).

The control site, which was not underneath the cable but was adjacent (DU4) or 12 ft from the street (DU5), demonstrated the important contribution of the roadway to surface lead levels. DU4, which could not have been significantly impacted by the suspended cable, had levels similar to DU1 (next to the roadway and underneath the cable), with the discrete sample measuring 222 ppm and the average of the three ISM samples of 252 ppm (range 180–316 ppm). DU5, which was ~12 ft from the roadway, had a discrete sample value of 88.5 ppm lead, and the average of the three ISM samples was the same, 88.5 ppm (range 80.9–94.9 ppm).

Samples collected 2–6 inches below the surface were generally similar to those seen in the 0–2 inch surface samples, but levels generally dropped to "background" (less than ~ 50 ppb) below 6 inches.

The results of this carefully conducted study demonstrate the important impact that roadways have on surface soil lead values. Levels adjacent to the roadway were generally in the 200–300 ppm range, whereas moving only 6–12 ft from the roadway resulted in levels less than 100 ppm. The study further demonstrated that the suspended aerial lead-clad cable had no discernable impact on soil lead values beneath the cable, as the control area adjacent to the same roadway (DU4) had surface soil lead levels nearly identical to that seen in DU1, situated the same distance from the roadway but immediately beneath the aerial cable.

j. Aerial lead-clad cable study, Denison, TX.

Ramboll also conducted a study in Dennison, TX. (Ramboll 2024b.) The study design was nearly identical to that described above for the Dallas, TX study, except that the roadway adjacent to the suspended cable was two lanes, rather than four, and it was located in an older residential neighborhood.

As in Dallas, both discrete and ISM sample methods were utilized. DU1 was, again, both adjacent to the roadway and underneath the cable; DU2 was adjacent to DU1 but further from the street; DU3 was adjacent to DU2 but further from the street; and DU4 and DU5 were control samples across the street (Figure 13). However, it should be noted that, after the sampling had been completed, it was learned that the "control site" had been compromised by the removal of a building and a gravel parking lot in 2019. Thus, the surface soil samples collected at the control site (DU4 and DU5) would not be expected to show the impact of years of lead deposition from automobiles traveling on the street.

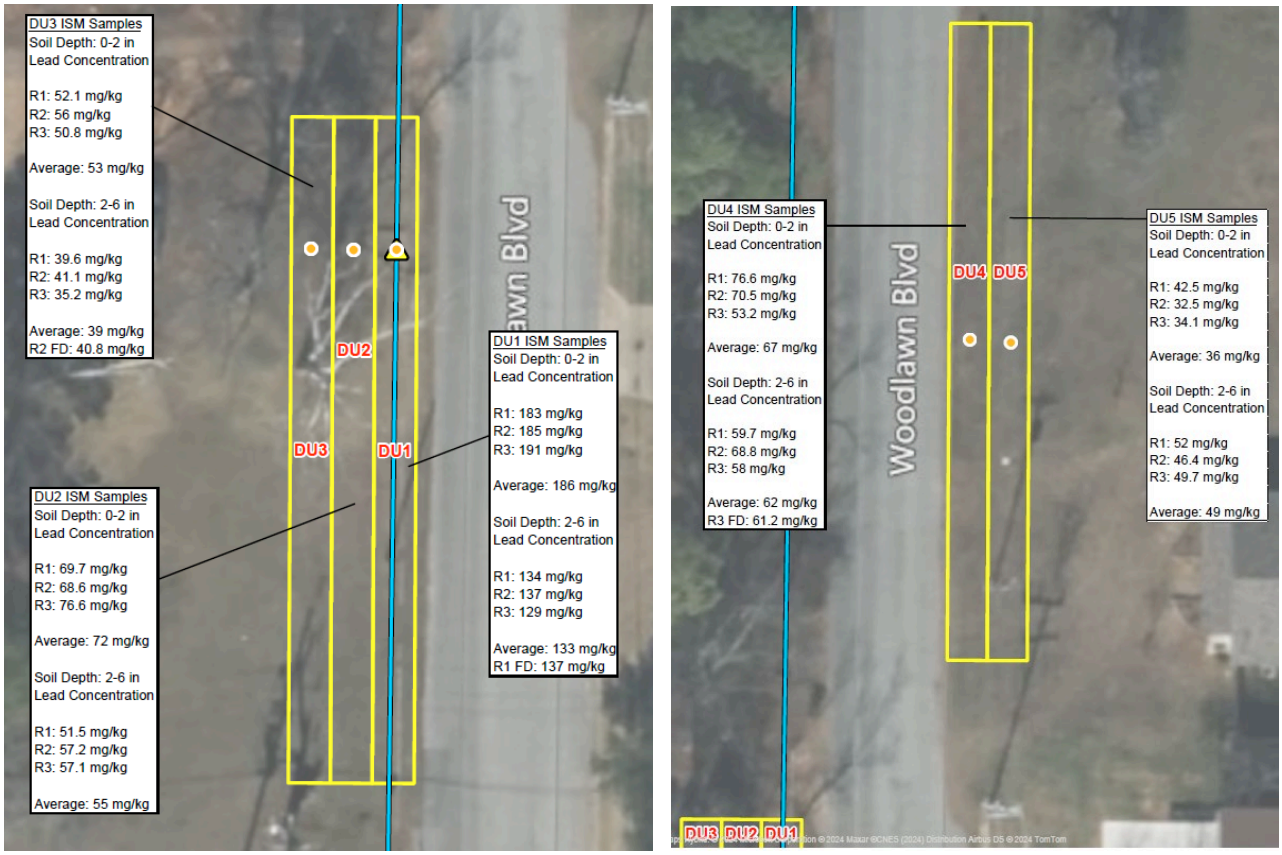


Figure 13. Soil sampling beneath Aerial Cable (left, DU1, DU2, DU3) and Control site (DU4, DU5) in Denison, TX. (From: Ramboll 2024b.)

As seen in Dallas, samples in DU1, underneath the cable and adjacent to the street, had lead levels somewhat higher (three surface samples average of 186 ppm), relative to levels in DU2, 12 feet from the street (average = 72 ppm) and DU3 (average = 53 ppm). DU4, adjacent to the street, had an average value of 67 ppm, and DU5 had an average of 36 ppm. Because of the removal of the gravel parking lot, which was in the immediate vicinity of both DU4 and DU5, in 2019, it is difficult to draw any significant conclusions from this control site. The control sample immediately adjacent to the street (DU4) did have lead levels approximately twice that of the control area 12 feet from the street (DU5), suggesting some impact from traffic-related lead deposition. Samples from DU4 were approximately three times lower than samples in DU1 (67 ppm, 186 ppm). Whether the suspended aerial cable contributed to the lead in soil at this site cannot be determined, given the uncertainty of the validity of the control sample adjacent to the roadway. Even if a portion of the lead found in surface soil in DU1 was from the aerial cable, the contribution, if any, is of no public health significance since the values in the soil are below the new US EPA RSL for residential areas of 200 ppm.

k. Summary of Soil Studies Beneath Aerial Lead-Clad Communications Cables.

Collectively, detailed and replicated analyses of soils have been conducted beneath seven different aerial telecom locations in the US: 1) Dearborn Heights, MI, 2) Trenton, MI, 3) Wappinger Falls, NY, 4) West Orange, NJ, 5) Coal Center and California Boroughs, PA, 6) Dallas, TX, and 7) Denison, TX. In several locations, multiple testing was completed by different contractors/organizations. Overall, the results suggest that, in a few instances, aerial cables may have had a modest impact on the levels of lead in soil beneath the cables, with levels typically less than twice the average background from adjacent sites. In several instances, no impacts from the cables could be discerned. It should be noted that in all sites, the soil lead concentrations beneath the cables were highly variable and generally ranged within the typical levels of lead in soil commonly seen in many urban environments, especially for sites close to roadways. Further testing of soils beneath aerial cables that are not in urban environments or close to roadways, with appropriate adjacent "background" samples away from the cables, would help to definitively answer this question.

Thus, if lead-clad telecom cables were suspended directly above children's play areas, it is possible that, over many years, such cables could contribute to existing urban background levels. If the area is covered with grass or gravel, possible exposure of children to lead in the underlying surface soil would be minimal. In all other areas (non-residential), the levels of lead in soil beneath aerial lead-clad cable seldom exceeded the US EPA RSL of 800 ppm lead in surface soil for non-residential areas. Because all the sampling conducted to date has been in urban areas next to roadways, it is currently impossible to determine the relative contribution of the various anthropogenic sources (e.g., deteriorating lead-paint dust, leaded-gasoline vehicle exhaust) from that which may have come from aerially suspended lead-clad cables.

2) Estimating Potential Public Health Risk from Exposure to Lead from Aerial Telecom Cables.

In the absence of any significant increase in soil lead levels above typical background levels from naturally occurring lead and other anthropogenic sources of lead, there is no need to conduct additional quantitative exposure estimates for the purposes of evaluating risk.

I conclude, with a reasonable degree of scientific certainty, that aerial lead-clad telecom cables may have a modest impact on lead in soils immediately beneath them, but the impact is well below additions to background that would exceed the screening level set by US EPA for non-residential areas (800 ppm). From the limited data available, it appears possible that small additions of lead to background soil could elevate lead levels in soil above the new US EPA RSL of 100–200 ppm for residential areas where children play (US EPA January 17, 2024). However, as noted above, if such areas are covered with grass, gravel, or other barrier to dust, the extent of exposure to lead-containing dust on a daily basis over years is highly unlikely. Thus, it is my opinion that, under the vast majority of circumstances, it is highly unlikely that aerially suspended lead-clad cables could contribute to exposures to lead sufficient to have any measurable adverse health impacts in children or adults, and thus, they present no significant

public health risk. My conclusions are consistent with those drawn by the New York State Department of Health and the US EPA, which drew site-specific conclusions for the specific sites discussed above (Wappinger Falls, NY and West Orange, NJ). I do note that this conclusion is based on limited information, and additional sampling of other sites would provide more confidence in the generalizability of this conclusion.

F. Potential Children's Exposure to Lead in Water from Submerged Telecom Cables.

The WSJ raised concerns about the presence of lead-clad cables that are submerged in bodies of water, such as Lake Tahoe, suggesting that the lead in cable sheathing may present a hazard to those using the water for recreational purposes. In order to assess whether there are any significant public health concerns related to exposure to lead derived from submerged lead-clad telecom cables, one needs information on two variables: (1) the concentration of lead in water in the immediate vicinity of the cable, from which one could estimate potential concentrations at the surface where swimmers and other recreational users might be exposed; and (2) the amount of water that might be consumed during swimming or other recreational use of the water. As noted above, there is substantial evidence that lead dissolved or suspended in water does not have any measurable impact on BLL via dermal absorption, so ingestion of water is the only viable pathway of exposure to lead in water.

1) Submerged Telecom Cable Exposure Assessment.

i. Concentration of Lead in Water Surrounding Submerged Telecom Cables.

When assessing the risk of exposure to lead from submerged lead-clad cables, the most important variable is the concentration of lead in water that might be consumed by a child swimming in a body of water with a submerged lead-clad telecom cable.

Lake Tahoe studies:

In a study conducted by Ramboll, seven water samples were collected immediately adjacent to two different submerged lead-clad telecom cables in Lake Tahoe in June 2023. (Ramboll 2023b.) Another eight samples were collected from control parts of the lake that could not have been impacted by the submerged cables. In the seven samples analyzed in water collected immediately adjacent to the cables, five were below the reporting limit for lead of 0.02 µg/L, and two were above. The two samples above the reporting limit of 0.02 µg/L were 0.044 and 0.064 µg/L. All the samples collected from the control areas were below the US EPA's reporting limit of 0.02 µg/L, and six of the eight samples were below the analytical detection limit of 0.006 µg/L. Those above the detection limit ranged from 0.010 to 0.018 µg/L.

These results are remarkably similar to those obtained from a previous water sampling study in Lake Tahoe in March 2021 by a different environmental consultant, Haley & Aldrich. Haley & Aldrich identified five locations for sampling lake water adjacent to and distant from the same

two submerged lead-clad telecom cables that were studied by Ramboll two years later. (Haley & Aldrich 2021.) Sampling Station 1 was selected because it was close to a submerged lead-clad telecom cable (Cable A), with the underlying lead sheathing in direct contact with lake water. Sampling Station 2 was selected because it was close to a second submerged lead-clad telecom cable (Cable B), which spanned the entire entrance to Emerald Bay in Lake Tahoe. In addition to these two sampling sites, three additional sites were selected at varying distances from the submerged telecom cables. Sampling Stations 3 and 4 were approximately 100 feet and 600 feet northeast of Station 1, respectively. Station 5 was a reference site near the Tahoe Keys area of South Lake Tahoe, approximately 3.5 miles southeast of the two submerged cables and Sampling Stations 1 and 2. Water samples at Sites 1 and 2 were collected at cable-depth near the shallow lake bottom, approximately four inches away from each cable and also approximately six inches above each cable (a duplicate sample was also collected at this depth). Additional water samples were collected midway between the lake surface and lake bottom, and approximately six inches below the lake surface. This last sample would be most reflective of water that might be ingested while swimming.

The results from the Haley & Aldrich (2021) study of the two submerged cables found that the lead concentration was below the detection limit (0.043 µg/L) in the samples collected nearest the cables (~four inches of each cable). Lead was not detected in most of the other samples collected from Stations 1 and 2, nor in most of the samples collected at the comparison and control locations away from the cables (Stations 3 through 5). In the two samples where lead was detected, the highest level measured (0.057 µg/L) was just above the detection limit.

Bayou Teche, Louisiana studies:

In a study conducted by the US EPA (Region 6), eight surface water samples in the vicinity of exposed lead-clad cables in Louisiana were collected and analyzed for lead. Only two of the eight had lead above the detection limit of 3.2 µg/L. These were shallow, stagnant water sources that would not be a source for recreational swimming, and thus exposure to such water would not occur to any significant extent. (US EPA 2023a, 2023b.)

A study on the potential water and sediment lead contamination in the Sugar Oaks Road site in the Bayou Teche, New Iberia region was completed by Ramboll in August of 2023. (Ramboll 2023f.) At this site, a lead-clad cable was identified as protruding from the water near the Bayou Teche shoreline and angled from downstream toward upstream. The study found that both dissolved lead and total lead concentrations in the surface waters were undetectable at all sample locations near or downstream from the cable.

A nearly identical study to the one above at a different site in the Bayou Teche area (Front Street/Bridge Street site) was also completed in August of 2023. (Ramboll 2023g.) Water samples were collected at six locations for the sampling area, including five stations near the cables and one remote reference location. All six samples had levels of dissolved lead below the detection limit. Unfiltered samples contained trace amounts of lead from suspended particulates, ranging from 0.93 to 4.75 µg/L. Two of the samples were above the method reporting limit of 2 µg/L but well below the drinking water standard of 15 µg/L.

An additional study of tidal surface water at the Bayou Teche-Franklin site in Louisiana was conducted by Ramboll (2023h). to determine the potential for the telecommunication cables in a tidally influenced shallow pond to contribute to lead concentrations in the water. Surface water samples were collected near the cable splice box and analyzed for lead. All analyses for dissolved lead were below the limit of detection of 0.6 ug/L. Small amounts of lead, slightly above the detection limit but below the limit of quantitation (ranging from 0.93–1.6 ug/L) were identified in an analysis of total lead that included suspended particulate matter, suggesting that small amounts of lead were present as particulate matter in the water. The highest concentration measured (1.6 ug/L) is only about 10% of the US EPA action level for lead in drinking water of 15 µg/L. The authors of the report also compared the results to historical assessments of lead in water in the Bayou Teche area and concluded, "*Based on historical data and our analysis of the sampling results, we conclude the water quality at the Site is not being adversely impacted by the cables and their splice box."* (Ramboll 2023h.)

Detroit River study:

An area along the Detroit River in Michigan was found to have seven submerged lead-clad cables emerging from the shoreline along the Trenton Channel of the Detroit River. It should be noted that the Trenton Channel and Detroit River are in a highly industrialized area and have been subject to multiple investigations and cleanups over time due to elevated contaminants of concern, including lead. Published research on lead pollution in the Detroit River have identified a variety of sources that may have introduced lead into the watershed. Ramboll collected 14 water and sediment samples along the area near the exposed lead-clad cables. Seven samples were collected downstream, and seven samples were collected upstream, along with two reference samples, at two different times (August 16 and August 28, 2023). Dissolved lead levels were below the limit of detection of 0.6 µg/L in all 32 samples, whereas in unfiltered samples, total lead was below the level of detection (0.22 µg/L) in 26 of the 32 samples. For the six samples above the detection limit, lead levels ranged from 0.23 to 0.62 µg/L. (Ramboll 2023i.)

As shown in Table 2, Caravanos et. al (2024) also measured lead in sediment surrounding submerged lead-clad cables in southern Louisiana. However, the paper provided insufficient details and no local "background" sediment samples in the vicinity. Thus, it is impossible to draw any conclusions from this study regarding the contribution of submerged lead-clad cables to levels of lead in the water surrounding the cables.

Thus, collectively, these studies provide strong support for the conclusion that submerged lead-clad cables contribute little, if any, lead to water in close contact with a submerged cable. It is obvious that the surface concentrations of lead that hypothetically could be derived from a submerged cable would be a very small fraction of any concentration measured immediately adjacent to the cable because of the enormous dilution that would occur as wave actions mix bottom waters with surface waters.

ii. **Consumption of Water During Recreational Activities.**

Several studies have measured and/or otherwise estimated the extent of water consumption during swimming. For example, Dorevitch et al. (2011) estimated the amount of water ingested during swimming in a pool, based on both self-reports and measurement of cyanuric acid (CYA) in urine. CYA is a metabolite of chlorine by-products used in water chlorination, so is a useful biomarker of ingested swimming pool water. They reported that ingestion of water from swimming was highly variable, with mean and upper confidence estimates of water ingestion during limited-contact recreation on surface waters of about 3–4 ml and 10–15 ml, respectively. These results are consistent with a more recent study by Dufour et al. (2017), which used similar techniques to determine estimates of water ingestion after swimming and other recreational activities. The results of the Dufour et al. (2017) study are shown in Table 3:

Table 3. Estimated Amounts of Ingested Water from Swimming. (From: Dufour et al. 2017.)

		Geometric means and 95% confidence intervals		
Age group	**Gender**	**Amount ingested (mL)**	**Time in the pool (mL per min)**	**Ingestion rate (mL per h)**
Children	All	38.2 (28–52)	95.9 (88–104)	*23.9 (17–33)*
Teens	All	22.1 (17–28)	55.8 (52–59	*23.7 (19–30)*
Adults	All	10.4 (9–12)	50.3 (49–52)	12.4 (11–14)
	Female	8 (7–10)	*51.1 (49–54)*	9.4 (8–11)
	Male	13.7 (11–17)	*49.5 (47–52)*	16.4 (13–20)

Differences between italic values are not significant. All other differences among age groups and gender are statistically significant at $p < 0.01$.

Thus, ingestion of 33 ml of water during a one-hour swim in a lake (the 95th-percentile value for children; the highest estimated intake group) would be a reasonable, risk-averse assumption for the purposes of exposure assessment.

2) Estimating Potential Public Health Risk from Exposure to Lead from Ingestion of Water in Recreational Waters Near Submerged Telecom Cables.

For the purpose of this risk assessment of the potential impact of lead derived from submerged lead-clad cables, I will assume an ingestion of 33 ml of water per recreational event for children, as discussed above. This represents an "upper bound" estimate. For concentration of lead in the water, I will use the maximum concentration of lead measured adjacent to the submerged lead-clad telecom cable (0.064 µg/L), recognizing that any lead released to the water immediately adjacent to the cable would be diluted many-fold before it reached the surface where swimmers might be exposed.

Based on these two upper-bound estimates for determining exposure, the maximum potential exposure to lead from submerged lead-clad cable would be 0.064 µg/L x 0.033 L or 0.002 µg of lead. This assumes that all the water ingested by a child who consumed 33 ml of water during an hour of swimming (the upper 95% Upper Confidence Limit [UCL] of consumption) contained the highest concentration of lead measured next to the submerged cable. As noted above, it would be physically impossible for this to occur, as the concentration of lead in water adjacent

to the submerged cable would be diluted by many orders of magnitude before reaching the surface where a swimmer might contact it. Thus, the actual amount of lead potentially ingested would be a small fraction of the 0.002 µg estimate. To put that number in perspective, the FDA established an "Interim Reference Level" of 3 µg/day of lead in the diet of children. The worst-case exposure scenario described above for exposure to lead from submerged lead-clad cables of 0.002 µg/day is 0.07% of the FDA's Interim Reference Level for dietary lead, which has been demonstrated to be protective of children's health, based on an extensive review of the existing data. (Flannery et al. 2020.)

The sediment study by Ramboll (2023c), discussed above in Sections III.D.3 and VI.D.1.i, demonstrated that the lead in submerged lead-clad cables in Lake Tahoe did not, to any significant extent, leach or migrate from the cables into surrounding sediments. This provides further evidence that submerged cables are not likely to be a significant source of lead in lakes or other bodies of water containing such cables.

Given the extremely low level of lead exposure that could occur from consumption of lake water near a submerged lead-clad telecom cable, I conclude, with a reasonable degree of scientific certainty, that submerged lead-clad cables are highly unlikely to contribute significantly to background lead exposure from all sources, and thus present no significant public health risk.

Finally, I note the WSJ reported much higher levels of lead adjacent to these same submerged cables in Lake Tahoe. The article and other information conveyed do not provide any significant details as to exactly how, or where, these samples were collected. If the sampling device used to collect the water sample was touching the cable or generated strong turbulence immediately adjacent to the surface of the cable, it is likely that the collected sample contained oxidized lead particles adhered to the surface of the cable. While this could account for relatively high levels of lead in water samples reported in the WSJ articles, it would not be representative of water levels of lead at the surface that might be consumed by swimmers or other recreational users of the lake.

G. Potential Children's Exposure to Lead in Soil from Surface-Exposed Buried Telecom Cables.

Buried lead-clad telecom cables can reach the ground surface at both the initiation and the end of the cable. Frequently the cables are joined in a "junction box," which itself may contain elevated levels of lead. Because both the lead-clad cable and the lead-containing junction box are at the surface and exposed to the elements, it could be expected that some lead could be released from either the exposed cables and junction box, or both. Since this is at the ground surface, the magnitude of potential releases of lead to surrounding soil requires a separate assessment. As discussed above, surface soil is subject to several important anthropogenic sources of lead (e.g., lead-based paint, tailpipe emission of lead, and industrial processes) and thus, it is accepted practice to identify lead in surface soil that is substantially above the typical levels of lead, which range from 10–200 ppm.

1) Surface-Exposed Buried Telecom Cable Exposure Assessment.

Because of lead soldering and other disturbances of lead-clad cables, exposed cables may contain relatively high concentrations of lead exposed to the elements, and thus consideration of the extent of soil lead exposure that could occur from a child playing in the proximity of surface-exposed cables should be considered, based on validated soil lead data where exposed cables and junction boxes are present.

i. ***Historical Data*.**

No historical data on lead in soil surrounding exposed lead-clad cables or splice junction boxes for the same was identified.

ii. ***Current Data*.**

Bayou Teche area

AT&T retained Exponent, Inc. (Exponent) and Geosyntec to conduct studies of five different lead-clad telecom cables in Louisiana, referred to as the Bayou Teche area. Although the specific locations of samples collected in the Bayou Teche area of Louisiana by the WSJ were not identified, I understand the Exponent and Geosyntec sample locations were intended to reflect, as near as possible, the same sites of exposed cables in the Bayou Teche area reported by the WSJ.

Site 1: The Exponent (2023) report provides: "*Site S-1 [was] the location of a severed telecom cable that extends above the ground surface on a slope leading down to Bayou Teche . . . The [X-Ray Fluorescence] reading for the cable was 54 percent lead. Three soil transects were sampled at 1 in., 3 in., 6 in., 12 in., 24 in. (2 ft), 48 in. (4 ft), and 96 in. (8 ft) distances away from the cable sheath in addition to a soil background location 162 in. (13.5 ft) up slope. Samples were collected at 0–1 in. and 1–3 in. depth intervals."* Samples were collected on three different transects, as shown in Figure 14.

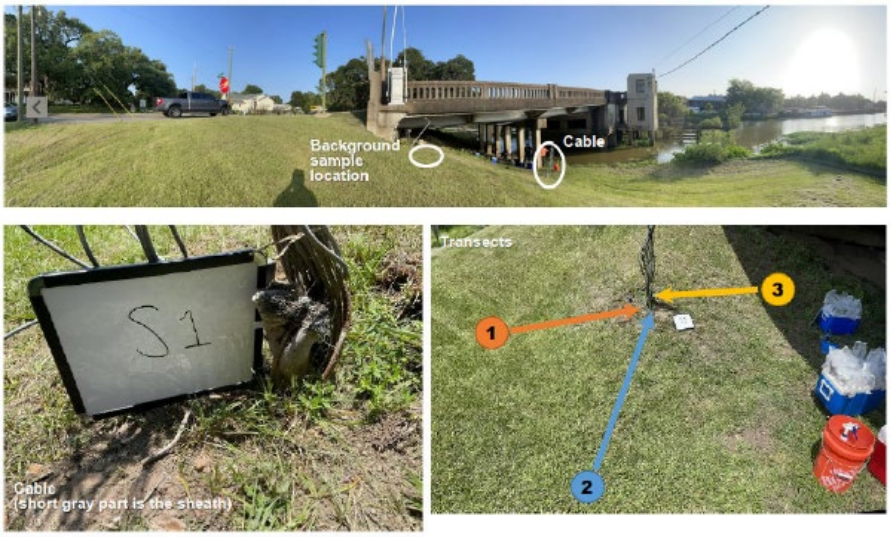


Figure 14. Site S-1 Annotated Photographs of Telecom Cable, Sample Transects, and Background Sample Locations. (From: Exponent 2023.)

The soil lead data for Site S-1 are shown in Table 4 below. It is evident from these data that surfaced-exposed lead-clad telecom cables, as expected, can undergo surface corrosion, to the extent that lead is actually released from the cable sheath into the soil immediately surrounding the cable. If the cable is frayed, it can release solid flakes of lead, as indicated in one sample from Transect 2 that contained 25,000 ppm lead (the sample likely contained a flake of lead sheathing, as additional testing of other samples from the same area yielded much lower results, in the range of 1,200 ppm to 1,900 ppm). In general, the level of lead in soil surrounding the exposed lead-clad cable decreased rapidly from ~1,000 to ~2,000 ppm immediately adjacent to the cable and similar to background levels within a few feet of the cable. Because the lead-contaminated soil is at or near the surface, rainwater likely moved the lead-contaminated soil particles on or near the surface of the cable through physical forces that would not likely be present in buried cable.

Table 4. Lead Results for Site S-1 Soil Samples. (From: Exponent 2023.)

Distance from Cable	Lead (mg/kg)			
	Transect 1	Transect 2	Transect 3	Background
0–1 in. depth interval				
1 in.	700	2,000	950	
3 in.	700	25,000/1,900[b]/1,200[b]	530	
6 in.	660	1,000	420	
12 in.	240	870	270	
24 in.	230	1,800/1,100[c]	230	
48 in.	170	210	210	
96 in.	68	90	250	
162 in.				150
1–3 in. depth interval				
1 in.	1,200	1,800	990	
3 in.	840	1,700	830	
6 in.	400	930	450	
12 in.	250	960	220	
24 in.	170	310	180/140[c]	
48 in.	180	220	160/130[c]	
96 in.	82	190	180/210[c]	
162 in.				180

Notes: [a] See Appendix B for data with laboratory qualifiers
[b] Additional aliquot of same soil sample digested and analyzed
[c] Field duplicate

This conclusion is supported by the fact that the distance of migration of lead from the cable was somewhat further in Transect 3, which is downhill, compared to Transects 1 and 2. Thus, these data show that these surface-exposed lead-clad cables did release small amounts of lead into the immediate environment of the cable but movement away from the cable is limited to distances of less than approximately eight feet. At distances beyond that, soil lead levels do not appear to be significantly impacted by releases of lead from the cable. Beyond four to six feet of the exposed cable, lead levels in the soil were near or below the US EPA's RSL of 200 mg/kg, although the Transect 2 "downhill" sample was above this level (~1,100 ppm) at two feet from the cable. However, the lead level in the sample taken at four feet was in the range of typical background urban soil samples.

Site S-3: The Exponent (2023) report provides that Site S-3 was a splice box that *"is partially exposed through the ground surface . . . The site is about 315 ft from a two-lane bridge with regular vehicular traffic. There is no general public access to this site, but there are other residential properties in the area. The splice box is on an angled bank sloped down towards Bayou Teche"* (Figure 15). An X-ray fluorescence reading determined that the splice box contained 1.3% lead. However, it was not possible to determine whether the cables inside of the splice box were lead-clad, as the cables were inaccessible.

Figure 15. Exponent Sampling site S-3 in Bayou Teche region, New Iberia, LA. (From: Exponent 2023.)

Sampling of soil was conducted as follows:

- Transect 1: 12 inches and 18 inches. Sample collection was limited to the 0–1 inch depth interval because debris restricted access to deeper depths.
- Transect 2: 48 inches (4 ft) and 72 inches (6 ft). Sample collection was limited to the 0–1 inch depth interval because debris restricted access to deeper depths.
- Transect 3: 1 inch, 3 inch, 6 inch, 12 inch, 36 inch (3 ft), and 72 inch (6 ft). Samples were collected at 0–1 inch and 1–3 inch intervals for distances of 12 inches, 36 inches (3 ft), and 72 (6 ft) inches, but only the 0–1 inch interval for distances of 1 inch, 3 inches, and 6 inches.
- A background soil sample was collected approximately 260 feet east from the splice box at 0–1 inch. In addition, a wipe sample of the splice box was collected.

All soil samples contained lead at background levels (<60 ppm in all samples).

Site S-4: A second splice box was also analyzed (Site S-4) at E. Bridge St, St. Martinsville, LA (Exponent 2023). That site contained a telecom splice box connected to a utility pole and the bare soil and grass between East Bridge Street and the sidewalk (Figure 16).

Figure 16. Annotated pictures from Exponent Site 4, Bridge St, St. Martinsville, LA. (From: Exponent 2023).

The splice box had three cables leading up from the ground into the box. The X-ray fluorescence reading for the splice box was 53% lead. Two soil transects were sampled at 0–1 inch and 1–3 inch depth intervals:

- Transect 1: 1 inch, 3 inches, 6 inches, 12 inches, 24 inches (2 ft), and 48 inches (4 ft).
- Transect 2: 1 inch, 3 inches, 6 inches, 12 inches, and 24 inches (2 ft).
- Background soil samples were collected from 0–1 inch and 1–3 inch depth intervals, 21.5 feet west of where the cables enter the ground.

The results from this site are shown in Table 5 below. The results demonstrate that some lead had been released into the soil in the immediate vicinity of the cables in the splice box. Soil samples taken one inch from the cables showed lead levels slightly in excess of 1,000 mg/kg,

declining with distance from the cable. At two feet and beyond, lead levels in soil were similar to background levels.

Table 5. Lead Results for Site S-4 Soil Samples. (From: Exponent 2023.)

Distance from Cable	Lead (mg/kg) Transect 1	Transect 2	Background
0–1 in. depth interval			
1 in.	1,300	1,500	
3 in.	550	430	
6 in.	420	210	
12 in.	270	150	
24 in.	190	200	
48 in.	160		
21.5 ft			170
1–3 in. depth interval			
1 in.	1,100	1,400	
3 in.	530	500	
6 in.	320	340	
12 in.	860	260	
24 in.	180/180 [b]	83	
48 in.	170		
21.5 ft			230

Notes: [a] See Appendix B for data with laboratory qualifiers
[b] Field duplicate

In a similar study, Geosyntec collected surface soil samples from a site on the southern/right bank of Bayou Teche (Figure 17). A lead-clad cable was observed loosely coiled up and lying on the bank of Bayou Teche (Site S-2). Sampling locations were designated along a transect at one inch, three inches, six inches, 12 inches, 24 inches (2 ft), 48 inches (4 ft), and 96 inches (8 ft) away from the base of the exposed cable. The results of the sampling at 0–1 inch from the surface are shown for all three transect samples, plus two samples collected within the coiled cable area are shown in Figure 17.

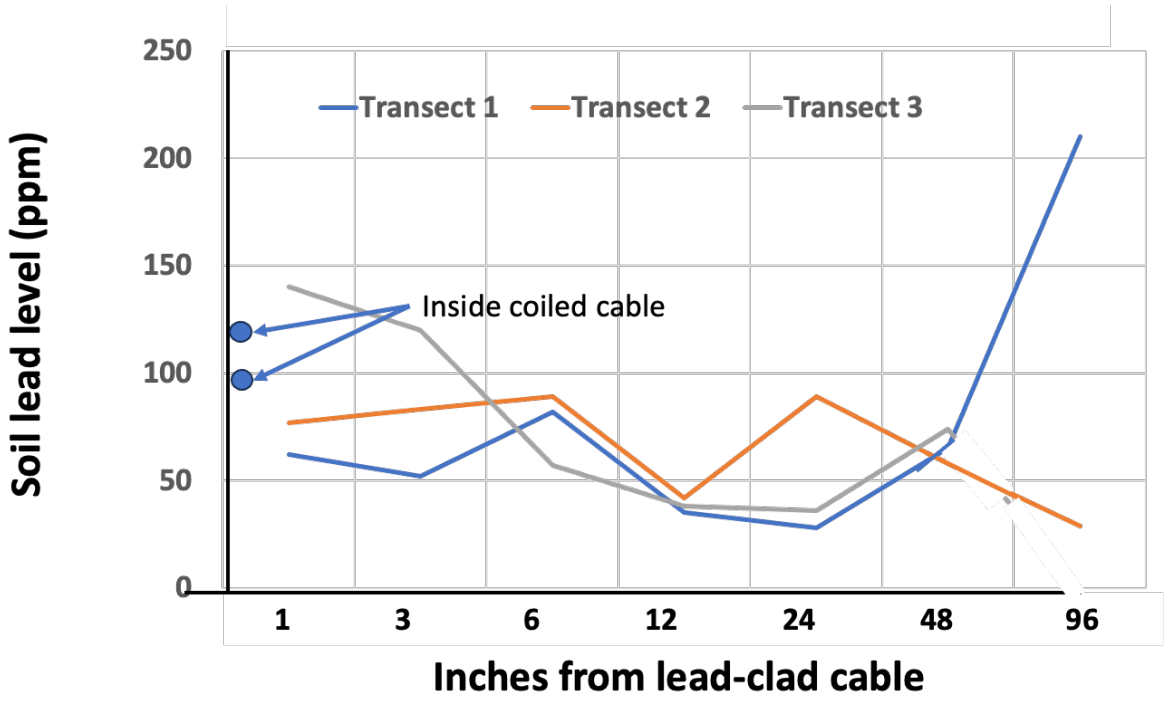


Figure 17. Surface Soil Concentrations of Lead Surrounding Exposed Telecom Cable at Bayou Teche. (From: Geosyntec 2023.)

Two points are evident from these data: (1) the concentrations of lead in the soil surrounding the cable were somewhat higher than the background levels taken 25 feet from the cable (28 ppm), but none were above typical urban background levels for lead in soil (100–200 ppm), and all but one were well below the US EPA's new RSL of 200 ppm lead for residential soil (one sample in Transect 1 was ~210 ppm, but it was 1 ft from the exposed cable, and thus is not likely to have come from the cable); and (2) if lead leaching from the lead-clad cable was a significant source of soil lead at distances away from the surface of the cable, one would expect a significant decrease in lead concentrations further away from the cable. It is evident from the data in Figure 17 that there is no significant gradient at any of the transects, demonstrating that the lead-clad cable is not contributing significantly to other anthropogenic sources of lead in surface soil in this location.

At Site S-5 (shallow pond) in the Bayou Teche area, Geosyntec collected sediment samples adjacent to a splice box/surface exposed cable. Geosyntec reported a lead concentration of 57 ppm in the sediment samples collected immediately next to the cable. The lead concentration decreased slightly to 33 ppm 12 inches from the cable. (Geosyntec 2023.)

In October 2023, Ramboll conducted an evaluation of lead release from surface-exposed cables near a floodwater wall in Bywater (a neighborhood within New Orleans), Louisiana. (Ramboll 2023d.) A brief description of the site, with figures, is provided in the report: "*A portion of lead-clad telecommunications cable is currently located in a utility conduit within the floodwall that runs parallel to the Mississippi River in the Bywater area of New Orleans, Louisiana. The utility*

conduit is in line with the intersection of North Peters Street and Port Street. The cable is severed where the conduit exits the concrete floodwall … The permit indicated that it was valid through 1962, which suggests the cable was installed sometime between June 1959 and December 1962. It is unclear when the cable was severed, as is the ultimate disposition of the portion of the cable formerly located outside of the floodwall." (See Ramboll 2023d report for figures illustrating the site.) Detailed inspection of the site suggested that the cable and surrounding areas had been disturbed, including evidence of fragmentation, suggesting efforts in the past of potential theft of copper from the lead-clad cable. Because of this, fragments of lead and the steel cladding were evident in the immediate vicinity of the exposed cable. Soil samples were collected from the area immediately surrounding the exposed lead-clad cable, as well as transect samples moving away from the exposed cable. The results of lead measurements are shown in Figure 18.

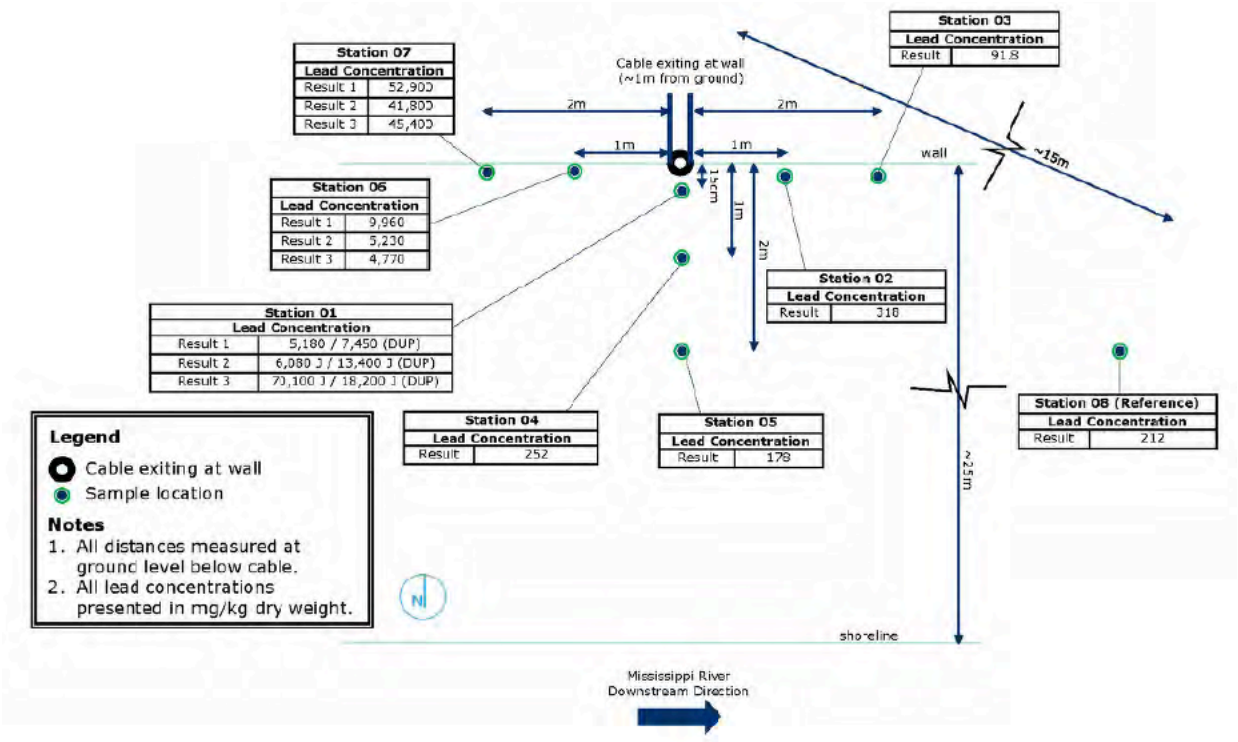


Figure 18. Soil lead sampling in the vicinity of an exposed lead-clad cable in Bywater, LA. (From: Ramboll 2023d.)

As indicated in Figure 18, fragments of lead were distributed in the immediate vicinity of the disturbed cable, giving rise to very high levels of lead (up to 70,000 ppm). However, lead in soil samples a few meters from the exposed cable were within the surface background levels of lead in this area (soil samples collected down river from the exposed cable ranged from 92–310 ppm, while a background sample taken away from the site was 212 ppm, consistent with background surface lead concentrations in urban environments.

A similar study was also done by Ramboll in October 2023, of lead release from surface-exposed cables near Donaldson, Louisiana. (Ramboll 2023j.) The location consisted of three lead-clad cables emerging from the ground into a cylindrical splice box, which was surrounded by a metal guard and plastic cover. Similar to the study conducted at the Bywater, LA exposed cable site, multiple soil samples were collected and analyzed in the vicinity of the exposed cables, as illustrated in Figure 19.

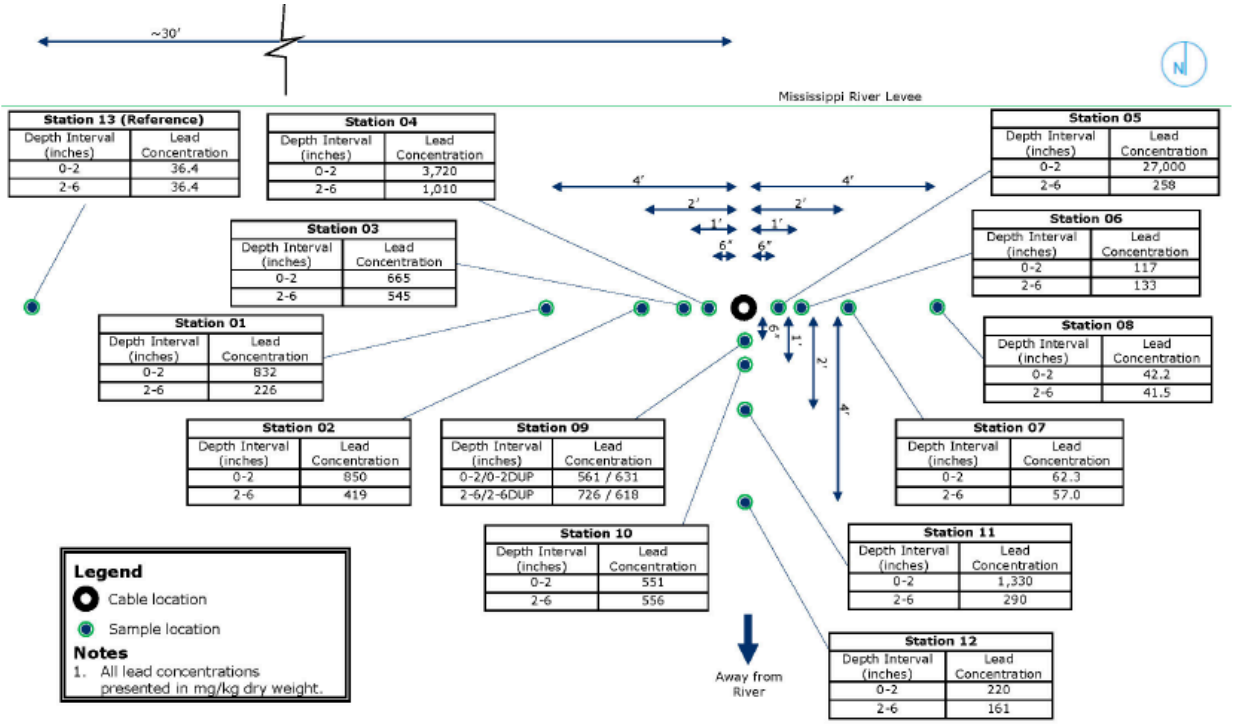


Figure 19. Soil lead sampling in the vicinity of an exposed lead-clad cable in Donaldsonville, LA. (From: Ramboll 2023j.)

In one sample immediately adjacent to the cable, a surface soil sample was found to have 27,000 ppm of lead, indicative of some deterioration and fragmentation of the lead cladding around the cable. Other samples within 1–2 ft of the cable also had elevated lead in the range of 600–3,720 ppm. One sample 2 ft from the exposed cable had a surface lead concentration of 1,330 ppm. Surface samples collected at 4 ft from the cable in various directions had lead concentrations of 42, 832 and 220 ppm lead. The reference sample was 36 ppm.

The results of the two Ramboll studies of exposed lead-clad cables in Louisiana demonstrate that surface-exposed lead-clad cables can release significant amounts of lead to soil in the immediate vicinity of the cable, but migration of the lead away from the cable is limited, such that soil levels more than 1–2 meters (3–6 ft) from the exposed cable appear to be less than the non-residential US EPA RSL of 800 ppm, but in some circumstances may be above the US EPA's new 200 ppm RSL for lead in soil in residential areas. However, none of the areas sampled here were in areas accessible to children.

Finally, in September of 2023 the US EPA Region 6 office undertook an exploratory survey of lead in soil, sediment, and water in six different Louisiana parishes (St. Martin, Iberia, St. Mary, Ascension, Assumption, St. Charles, and Orleans), where lead-clad cables appeared to be exposed at the surface. (US EPA 2023a, 2023b.) Although the report was limited in scope, the US EPA Region 6 reported on surface soil lead levels in 10 surface samples collected at five different sites (soil samples were not collected at St. Mary parish; duplicate samples were collected at 2 sites). The values ranged from 59–1,170 ppm. A November 2023 update to this report from the US EPA summarized the results of the study as follows: "*The results do show that some soil samples have lead concentrations above an EPA screening level of 400 parts per million. EPA's assessment of the data takes into account that most sample locations are in isolated areas where people are unlikely to congregate. Most areas are also covered with grass, which provides a natural barrier to reduce exposure.*" (US EPA 2023a, 2023b.)

Taken together, the results from all 11 different exposed cable sampling sites support the conclusion that release of lead from the cables generally does not extend beyond approximately two meters from the cables. However, disturbance of the lead cladding can, in some instances, result in very high levels in the areas immediately adjacent to the cable. If such areas are readily accessible to children, remediation of the exposed cables may be warranted.

2) Estimating Potential Public Health Risk from Exposure to Lead from Surface-Exposed Buried Telecom Cables and/or Junction Boxes.

As discussed above, whether lead contamination of soil constitutes a significant public health risk depends on three factors: (1) the concentration of lead in the soil; (2) how much and how frequently that soil is ingested; and (3) how much of the ingested lead is absorbed into the bloodstream (bioavailability). Based on the evaluation of lead in the soil at the Bayou Teche Site S-1, supported by other sites discussed above, the maximum concentration of lead at a distance greater than two feet from the exposed cables and junction box from one of the three transects (Transect 3) was ~1,500 ppm, whereas the lead concentration in the soil, two feet from the cable at both other transects at the same site was ~230 ppm. This level is similar to the soil background level at that site (~150 ppm) and the US EPA's RSL for residential areas of 200 ppm. The average concentration of lead in soil at two feet from the cable was 960 ppm. This value is slightly above the 800 ppm US EPA RSL for non-residential soils but again was limited to soils within 2 ft of the cable, which greatly limits the opportunity for exposure.

The second most important variable to be considered is the amount of this soil that could be reasonably expected to be ingested by a child frequenting the area near the cables. As noted above, the US EPA's estimated daily soil ingestion rates for children (1–8 years old) averages about 70 mg/day (ranging from 50 to 90 mg/day, depending on age; see Table 1). Studies have demonstrated that slightly more than half of this ingestion comes from indoor house dust. Thus, one could consider an upper-bound estimated daily ingestion of total outdoor soil to be approximately 35 mg/day.

The third-most important variable is the fraction of daily outdoor soil that could have come from an area 2–4 feet surrounding the cable. Most US EPA models of lead in soil assume that

indoor dust contributes 40–60% of total daily soil/dust intake, with the remaining 40–60% coming from a combination of residential soil (e.g., backyards, gardens) and "community" sources. The US EPA's new screening value for residential areas is now 200 ppm (also presented in alternate units as 0.200 µg/mg). If a child ingests ~70 mg of soil/day, and ~50% of that is from outdoor soil, the amount of lead ingested would be about 14 µg per day. It follows then that any source of lead that potentially contributes to surface soil would need to elevate the overall intake of lead to a level significantly above ~14 µg of daily lead intake from all soil (70 mg of soil intake/day x 0.2 µg of lead/mg of soil = 14 µg/day).

As an example, assume, as a "worst case," that a child spends an average of 10% of their daily outdoor playtime close to lead-clad cable with increased surface soil lead level of 600 ppm. If 50% of their soil intake of 70 mg comes from indoor house dust and the other 50% comes from outdoor soils, then total outdoor soil exposure would be 35 mg. If 10% of the outdoor soil (3.5 mg) came from soil close to a lead-clad cable that contained 600 ppm lead (0.6 µg/mg soil), the child's exposure from the contaminated area would be ~2.1 µg of lead, of which ~67% (~1.4 µg) came from the lead-clad cable, if the urban background level was 200 ppm. If the indoor dust and outdoor soil contained the US EPA's new RSL of 200 ppm for lead (which is typical of urban background levels), the total daily exposure would be 14 µg of lead. Thus, this worst-case analysis suggests that the additional lead exposure from outdoor soil would contribute to an increase in exposure from soil of about 10%. According to the US EPA exposure model for lead in soil, an increased daily intake of 1 µg of lead would result in an increased BLL of 0.16 µg/dL if it occurred on a daily basis for months to years. As discussed previously (see Figure 2), a change in BLL of that size would not have any discernable impact on IQ/neurodevelopment using standard assessment tools used to evaluate IQ in children.

Based on these considerations, I conclude, to a reasonable degree of scientific certainty, that surface-exposed lead-clad telecom cables, as represented by data from 11 sites in different areas in Louisiana, will not add significantly to background exposures of children to lead, and thus present no significant public health risk. However, the data from several of the 11 sites indicates that lead from surface-exposed telecom cables can migrate to soil immediately surrounding the cables/junction boxes at levels exceeding US EPA's soil lead screening level of 200 ppm, and thus, it would be prudent public health practice to identify and secure such sites to minimize contact with affected soils in close proximity to the exposed cables.

VI. Conclusion.

Based on my decades of experience, review of the literature and recent validated data, and utilizing conservative "risk-adverse" assumptions, I conclude that lead-clad telecom cables present no significant risk to public health.

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Ramboll. Detroit River Water & Sediment Study, Detroit, MI, September 20, 2023i.

Ramboll. Donaldsonville Site Soil Lead Study, Donaldsonville, LA, October 27, 2023j.

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APPENDIX 1

CURRICULUM VITAE

1. BIOGRAPHICAL INFORMATION

DAVID L. EATON, PhD, DABT, FATS

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2. EDUCATION

1978	University of Kansas Medical Center, Kansas City, KS, **PhD,** (Pharmacology)
1974	Montana State University Bozeman, Montana, **BSc,** (Pre-medicine)

3. LICENSURE / CERTIFICATION

Diplomate of the American Board of Toxicology, 1981, recertified 1985, 1990, 1995, 2000, 2005, 2010, 2015, 2020

Fellow, Academy of Toxicological Sciences, 2000-present

4. PROFESSIONAL POSITIONS

12/07/18 – present	**Adjunct Professor of Pharmacology and Toxicology,** College of Pharmacy, University of Arizona
03/15/2019-present	**Emeritus Professor of Environmental and Occupational Health Sciences**, University of Washington
3/15/2013-3/15/2018	**Dean and Vice Provost,** The Graduate School, University of Washington
10/2010-7/2011	**Interim Vice Provost for Research,** University of Washington, Seattle, WA
2006-2013	**Associate Vice Provost for Research,** University of Washington, Seattle, WA
2003-2019	**Adjunct Professor,** Department of Medicinal Chemistry, School of Pharmacy, University of Washington, Seattle, WA
2000-2019	**Affiliate Member**, Public Health Sciences Division, Fred Hutchinson Cancer Research Center, Seattle, WA

1998-2019	**Professor,** Public Health Genetics Program (UIF), School of Public Health & Community Medicine, University of Washington
1995-2015	**Director,** Center for Ecogenetics and Environmental Health, University of Washington
1992-2019	**Professor,** Department of Environmental and Occupational Health Sciences, School of Public Health and Community Medicine, University of Washington
2003-2007	**Associate Director,** Fred Hutchinson Cancer Research Center / University of Washington / Childrens' Hospital and Medical Center *Cancer Center Research Consortium* (FHCRC/UW/CHMC CCRC)
1999-2005	**Associate Dean for Research**, School of Public Health and Community Medicine, University of Washington
1991-1993	**Associate Chairman,** Department of Environmental Health, School of Public Health and Community Medicine, University of Washington
1986-1991	**Director,** Toxicology Program, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington
1986-1992	**Associate Professor,** Department of Environmental Health, School of Public Health and Community Medicine, and Institute for Environmental Studies, College of Arts and Sciences, University of Washington
1979-1986	**Assistant Professor,** Department of Environmental Health, School of Public Health and Community Medicine, and Institute for Environmental Studies, College of Arts and Sciences, University of Washington
1978-1979	**Postdoctoral Fellow** in Toxicology, Department of Pharmacology, University of Kansas Medical Center, Kansas City, KS
1974-1978	**Predoctoral Trainee,** US Public Health Service, Department of Pharmacology, University of Kansas Medical Center, Kansas City, KS
1974 MT	**Research Assistant,** Dept. Chemistry, Montana State University, Bozeman,

5. HONORS, AWARDS, FELLOWSHIPS,

Merit Award, Society of Toxicology, 2024
David Rall Medal, National Academy of Medicine, 2020
Excellence in Pharmacology/Toxicology Lifetime Award, PhRMA Foundation, 2015
Public Communications Award, Society of Toxicology, 2014
PANWAT Achievement Award, Pacific Northwest Society of Toxicology, 2014
Elected Member, Institute of Medicine (now National Academy of Medicine), National Academies of Science Engineering and Medicine, 2011
Elected Fellow, Washington State Academy of Sciences, 2011
National Associate, National Academy of Sciences, selected 2004 (lifetime membership)
Fellow (Elected), Academy of Toxicological Sciences, 2000
Fellow, International Union Against Cancer, 1996

Fellow (elected), American Association for the Advancement of Science, 1995
Zeneca Traveling Lectureship Award (Society of Toxicology), 1995
Achievement Award, Society of Toxicology, 1993
Rohm & Haas Distinguished Professor of Public Health Sciences, 1992-97
NIH Pharmacology Research Associate Program Postdoctoral Fellowship Award, 1979-1981 (declined)

6. PROFESSIONAL ACTIVITIES (Outside of UW)

A. Professional Organizations

Society of Toxicology, 1982-Present
International Society for the Study of Xenobiotics (ISSX), 1992-2019
American Association for Cancer Research, 1988 – present
Molecular Epidemiology Group, 1999-2018
Sigma Xi 1987-present
Pacific Northwest Association of Toxicologists (Northwest Chapter, SOT) 1984-2019
Mountain-west Association of Toxicologists, 2019-present
American Association for the Advancement of Science, 1977-present

B. Scientific Advisory Boards and Panels - National

1. Service to the National Academies of Sciences, Engineering and Medicine

Review Monitor, "*Electric Arc Furnace Slag: Understanding Human Health Risks from Unencapsulated Uses*.", National Academy of Sciences, Engineering and Medicine / National Research Council, 2023.

Review Monitor, "*Advancing the Framework for Assessing Causality of Health and Welfare Effects to Inform National Ambient Air Quality Standard Reviews",* National Academy of Sciences, Engineering and Medicine / National Research Council, 2022.

Review Monitor, "*Meeting the Respiratory Protection Needs of the Public and Workers without Workplace Respiratory Protection Plans",* National Academy of Sciences, Engineering and Medicine / National Research Council, 2021-2022.

Review Monitor, *The Use of Systematic Review in EPA's Toxic substances Control Act Risk Evaluations,* National Academy of Sciences, Engineering and Medicine / National Research Council, 2021**.**

Review Monitor, *Compounded Topical Pain Creams: Review of Select Ingredients for Safety, Effectiveness, and Use* (2020), National Academy of Sciences, Engineering and Medicine / National Research Council, 2020.

Review Coordinator, *Review of Report and Approach to Evaluating Long-Term Health Effect in Army Test Subjects*", National Academy of Sciences, Engineering and Medicine / National Research Council, 2018.

Review Coordinator, *The Clinical Utility of Compounded Bioidentical Hormone Therapy,* National Academy of Sciences, Engineering and Medicine / National Research Council, 2018.

Reviewer, NASEM report *Review of Advances Made to the IRIS Process*, March 2018.

Chair, NAS/NAM/NRC, Committee on the Review of the Health Effects of Electronic Nicotine Delivery Systems, 2017-2018.

Review Coordinator, *Protocols for Conducting Systematic Reviews of Selected Endocrine-Disrupting Chemicals,* National Academy of Sciences, Engineering and Medicine/National Research Council, 2016.
Review Coordinator, Styrene Assessment in the NTP 12th Report on Carcinogens, National Research Council, 2014-15
Member, NAS/NRC Committee to Review the EPA IRIS process, 2012-2014.
Member, NAS/NRC Committee on the Future of Science at the EPA, 2011-2012.
Member, NAS/ Institute of Medicine committee on **"**Breast Cancer and the Environment: The Scientific Evidence, Research Methodology, and Future Directions", 2010-2012.
Review Coordinator, NAS/IOM/NRC NRC Study on Toxicity Pathway-Based Risk Assessment: Preparing for a Paradigm Change. 2010.
Review Coordinator, NAS/IOM/NRC, Review of EPA's Draft IRIS Assessment of Tetrachloroethylene (BEST-K-06-03-A), 2009 – 2010.
Chair, NAS/NRC Committee for Review of the Federal Strategy to Address Environmental, Health, and Safety Research Needs for Engineered Nanoscale Materials, 2008
Chair, NAS/NRC IOM Committee on Assessment of the Health Implications of Exposure to Dioxins, 2004-06.
Member, Working Group on Gene-Environmental Interactions, National Children's Study, NIH, 2001-04.
Chair, NAS/NRC Committee on Emerging Issues and Data on Environmental Contaminants, 2002-2005
Member, NAS/NRC Panel on Arsenic in Drinking Water, 2001
Liaison, NRC/BEST Subcommittee on Arsenic in Drinking Water, 1997-98
Member, Board of Environmental Studies and Toxicology, National Academy of Sciences/National Research Council, 1996-99

2. Service to the National Institutes of Health and other US Governmental Agencies

Special Advisor to the Director, National Institute of Environmental Health Sciences and National Toxicology Program, 2023-24
Chair, Board of Scientific Councilors Working Group on review of the draft "*NTP State of The Science Report on Fluoride*" and NTP Draft manuscript on *"Systematic Review and Meta-Analysis of Fluoride and Children's IQ",* 2023.
Chair, Board of Scientific Councilors, National Toxicology Program, 2020-2022
Member, Board of Scientific Councilors, National Toxicology Program, 2019-2022
Chair, National Toxicology Program Peer Review Panel, NTP Technical Reports on Cell Phone Radiofrequency Radiation Studies. March 21-23, 2018, NIEHS, RTP, NC.
Member, Search Committee, Director of the Division of the National Toxicology Program (NTP), NIEHS, Research Triangle Park, NC, October, 2016-2017.
Member- National Advisory Environmental Health Sciences Council (NIH/NIEHS Council), 2013-2017.
Member, External Science Advisory Board, Fundamental & Computational Sciences Directorate, Pacific Northwest National Laboratory, Richland, WA; 2011-2015.
Member, Search Committee, NIEHS Extramural Program Director, 2008
Member, NIH/NIEHS Special Emphasis Panel, ONES program review, 2007
Member, external review team for the National Center for Toxicogenomics, NIEHS, 2005.

***Ad hoc* member**, NIH Study Section, Epidemiology of Cancer-2 (EDC-2), June, 2003, March 2004
Member, NIH Center for Scientific Review, Digestive Disease Boundary Review Panel, 2002.
Member, Scientific Review Panel, Agricultural Health and Safety Centers, NIOSH, 2001.
External Reviewer, Environmental Health Sciences Initiative, Battelle Pacific Northwest National laboratory, Richland, WA, 1999-2000
Member, Environmental Genome Working Group, NIEHS, 1997-98
Member, NIEHS Site Visit Team and Reviewer, Program Project Grant review, Oregon State University, 1992, 1993.
Invited Participant, United States - Japan Cooperative Program on Development & Utilization of Natural Resources, Joint panel on toxic microorganisms symposium on "Cellular and Molecular Mode of Action of Selected Microbial Toxins in Foods and Feeds," National 4-H Center, Chevy Chase, MD, Oct. 31- Nov. 2, 1989
Invited Participant, Advisory workshop on significance and utilization of SARA Title III data, sponsored by USEPA, Chemical Manufacturer's Association and the National Academy of Sciences, NAS Headquarters, Washington, DC, Oct. 18-19, 1988
Member, NIH *Ad Hoc* Scientific Review Panel for NIEHS contract on "Methods development for *in vitro* human metabolism of xenobiotics," February 19-21, 1985, Research Triangle Park, NC

3. Service to Professional and Non-profit Scientific Organizations

A. Toxicology Organizations

Vice-President, Academy of Toxicological Sciences, 2024-25 (President in 2025-26)
Member, Society of Toxicology Foundation Endowment Board, 2015-2017
Chair, Celebrating Member Accomplishments Task Force, Society of Toxicology, 2011-2014
Member, Planning Committee, *Future of Toxicology II* Symposium, Society of Toxicology, 2013-14
Chair, Audit Committee, Society of Toxicology, 2012 (member, 2010-11).
Member (elected)**,** Nominating Committee, Society of Toxicology, 2005-06; 2007-08
Member, 50th Anniversary Planning Task Force, Society of Toxicology, 2007-2009
Board of Trustees, Academy of Toxicological Sciences, 2005-08
Chair, Society of Toxicology Task Force on NIH Grant Reviews, 2004-2007
Board of Directors, Toxicology Education Foundation, 2004-08; **Vice President**, 2005-06
Member, Board of Publication, Society of Toxicology, 2001-02
Member, Society of Toxicology Task Force on Professional Society Liaison Roles, 2004.
Member, Finance Committee, Society of Toxicology, 2001-02
Member, AAAS Education Sub-Committee, *Court Appointed Scientific Experts (CASE)*, 2000-01
Councilor, Mechanisms Specialty Section, Society of Toxicology, 1997-99
Secretary, Society of Toxicology, 1996-98 **(**Secretary-elect, Society of Toxicology, 1995-96)
Editor, SOT Communiqué, 1997-98
President, Society of Toxicology, 5/1/01-4/30/02 (Past President, 5/1/02-4/30/03; Vice-President, 5/1/00-4/30/01, Vice-President-elect, 5/1/99-4/30/00).
Councilor, International Union of Toxicology (IUTOX), 2001-04
President, Mechanisms Specialty Section, Society of Toxicology, 1996-97
Treasurer, American Board of Toxicology, Inc., 1991-94
Councilor, Mechanisms Specialty Section, Society of Toxicology, 1990-93
Chairman, Membership Committee, Society of Toxicology, 1992-93

Membership Committee, (elected committee), Society of Toxicology, 1990-93
Board of Directors, American Board of Toxicology, Inc., 1990-94
Member, Executive Committee, International Congress on Toxicology VII, 1993
Chairman, Local Arrangements Committee, International Congress on Toxicology VII, 1992
Committee on Public Communications, Society of Toxicology, 1984-1985
Member, Society of Toxicology *ad hoc* "Tox-90's Education Committee," 1988-1991

B. Other Environmental Health Sciences Organizations

Treasurer, Health and Environmental Sciences Institute (HESI), 2020-present
Member, Board of Trustees, Health and Environmental Sciences Institute (HESI), 2018-present
Member, Board of Trustees, Health and Environmental Sciences Institute (HESI) 2014-2015.
Chair, Research Committee, Health Effects Institute, Boston, MA, 2010-2018
Participant, 1987 Gordon Research Conference on Mechanisms of Toxicity, Kimball Union Academy, Meriden, New Hampshire, July 27-31, 1987

C. *Cancer Research Organizations*

Member, Molecular Epidemiology Working Group Program Committee, American Association for Cancer Research (AACR), 2007
Organizing Committee, International Conference on Molecular and Genetic Epidemiology of Cancer, AACR- Molecular Epidemiology group, 2002-03
Visiting Scientist, International Agency for Research on Cancer (IARC), Lyon, France, 9/1/95-12/1/96.

D. Editorial Boards & Grant Review

Editorial Board, *Chemico-Biological Interactions,* 1998-2012;
Toxicology Open journal, 2007-2009
External Reviewer, Cancer Research-United Kingdom; review of Cancer Center at University of Dundee, Scotland, June 17-18, 2010
Associate Editor, *Toxicology and Applied Pharmacology,* 1996-99
Editorial Board, Oncology Reports, 1994-97
Editorial Review Board, *Environmental Health Perspectives,* 1993-97.
External Grant Reviewer, Hong Kong Research Grants Council, University and Polytechnic Grants Committee, Hong Kong, 1992-93, 95-96, 99
Editorial Board, *Environmental Carcinogenesis & Ecotoxicology Reviews,* 1992-present
Board of Publications, *Society of Environmental Toxicology and Chemistry,* 1989-91
Editorial Board, *Toxicology and Applied Pharmacology,* 1989-1996

4. Service to other Academic Institutions

External Reviewer, O'Donnell Award, Texas Academy of Medicine, Engineering, Science and Technology (TAMEST), 2021-2023
Member, External Science Advisory Board, University of Arizona Southwest Environmental Health Sciences Center, 2019-2022.
Chair, External Scientific Advisory Committee, Texas A&M Superfund Research Program, 2019-present.
Member, External Scientific Advisory Committee, Texas A&M NIEHS T32 Training Grant, 2018-present.

Member, University of North Carolina-Chapel Hill Gillings School of Public Health, External Advisory Committee, 2014-2018
Member, Governmental and Regulatory Affairs Committee, Council of Graduate Schools, Washington, DC 2012-2018
Member, External Science Advisory Board, Center for Research on Environmental Disease, Texas A&M University/Baylor College of Medicine, Houston, TX, 2014-2016.
Member, External Science Advisory Board, University of Montana Environmental Health Sciences Center, Missoula, MT, 2003-2008
Member, External Science Advisory Board, Harvard University Environmental Health Sciences Center, 2003- 2014
Chair, External Science Advisory Board, NCRR Institutional Development Award, University of Alaska BRIN/INBRE program, 2001-2013
Member, External Science Advisory Board, Center for Research on Environmental Disease, University of Texas-MD Anderson Cancer Center, Smithville, TX, 1998-2011.
Member, External Science Advisory Board, University of New Mexico Environmental Health Sciences Center, 2003-2005.
Member, External Science Advisory Board, NIEHS Center for Environmental Health Sciences, University of California-Davis, 1999-2005
Member, External Science Advisory Board, NIEHS Center for Rural Environmental Health, Texas A&M University, College Station, TX, 2001-2005
Reviewer, Cancer Research –United Kingdom program grant, University of Dundee, Scotland, 2004
Member, Science Advisory Board, The Institute for Wildlife and Environmental Toxicology, Clemson University, Clemson, SC. 1990-94; Chairman, 1993.

5. Service to Industry

Member, External Scientific Advisory Board, Semiconductor Industry Association, 2005-2009
Chair, External Science Advisory Board, Procter and Gamble, Central Product Safety Division, Miami Valley Laboratories, Cincinnati, OH, 2003-2012
Member, External Science Advisory Committee, CIIT Centers for Health Research, 2005-2006
Advisory Board, BSCS, Inc, (Biological Sciences Curriculum Study), Colorado Springs, CO, 1999-2000

C. Scientific Advisory Boards and Panels – Community and State (excluding UW)

Chair, Membership Committee, Washington State Academy of Sciences, 2017-18
Co-Chair, Membership Committee, Washington State Academy of Sciences, 2016-17
Chair, Membership Committee, Section 4, Washington State Academy of Sciences, 2013-15
Member, Board of Directors, Washington State Academy of Sciences, 2012-18
Member, Board of Directors, Global Health Research Fund, (Gubernatorial appointment) 2011-2015
Member, Board of Directors, CARE Northwest, 1998-2008
Chair, Tobacco Control Advisory Committee, American Lung Association of Washington, 2002-2007
Member, Grant Advisory Committee, American Lung Association of Washington, 2003-2006
Member, Board of Trustees, Seattle Biomedical Research Institute (SBRI), 2004-2006

Member, Tobacco Control Advisory Committee, American Lung Association of Washington, 2000-02

Member, Advisory Committee for Risk Management Conference, Washington Foundation for the Environment, 1992-93.

Pacific Northwest Association of Toxicologists (Northwest Chapter, SOT); founding member, 1984-present, Vice President, 1984-1985; President, 1987-88, Councilor, 1988-89; Councilor, 1997-98

Member, Scientific Advisory Board, State of Washington Initiative For Chemically-Related Illness, Washington State Department of Labor and Industries, 1997

Member, Northwest Consumer Food Safety Council, 1990-94

Chairman, Science Advisory Board (5 member board established by Initiative, pre-empting above SAB), Washington State Department of Ecology, 1989-1991; Member 1989-92.

Member, Science Advisory Board (14 member board established under legislative act), Washington State Department of Ecology, 1988-90

Member, Technical Work Group on Environmental/Regulatory Policy for the Puget Sound Water Quality Authority, 1987

Site Visitor, Western Washington University, Bellingham, WA; Requested to evaluate current occupational health and safety practices relating to toxic chemical storage and use at Western Washington University. Provided written report to Dean of the Graduate School, October 8, 1986

Member, Mayor's Advisory Task Force on PCBs, 1984-86

Member, Advisory Task Force to Assess Health Impacts of Sediment and Shellfish pollution at Eagle Harbor, WA. For Coalition of County and State Health Departments. October 1984-85

Member, Mayor's Health Advisory Panel on Gas Works Park, 1984

Member, Gypsy Moth Advisory Committee, Department of Agriculture, Washington State Medical Association, and Office of the Mayor, City of Seattle, 1983

Member, Emergency Insect Criteria Committee, State Department of Agriculture, 1982-86

Member, Washington State Pesticide Advisory Board (Gubernatorial Appointment), 1982-1985, reappointed 1986-1989

Member, State Department of Social and Health Services, *Ad Hoc* Committee to Review Health Implications of Totally Recycled Water Systems, 1981

D. Manuscript Referee:

Aquatic Toxicology; Archives of Environmental Contamination and Toxicology; Biochemical Pharmacology; Biochemistry; ; Cancer Epidemiology, Biomarkers & Prevention; Cancer Research; Carcinogenesis; Chemico-Biological Interactions; Drug Metabolism & Disposition; Environmental Science and Technology; *Fundamental and Applied Toxicology; Journal of Pharmacology and Experimental Therapeutics; Molecular Pharmacology Nature Reviews-Cancer; Proceedings of the National Academy of Sciences; Pharmacology & Toxicology; Toxicology and Applied Pharmacology; Toxicological Sciences; Regulatory Pharmacology & Toxicology.*

7. BIBLIOGRAPHY

(*Indicates students, post-doctoral fellows or research technologists under the direction of Dr. Eaton).

A. ORIGINAL RESEARCH PAPERS IN REFEREED JOURNALS
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1. **Eaton DL,** Klaassen CD. Effects of acute administration of taurocholic and taurocheneodeoxycholic acid on biliary lipid excretion. *Proc Soc Exp Biol Med* 151:198-202, 1975.
2. **Eaton DL,** Poisner AM. Plasma pseudorenin in rats after alteration in the renin-angiotensin system. *Proc Soc Exp Biol Med* 154:6-8, 1977.
3. **Eaton DL,** Klaassen CD. Carrier-mediated transport of ouabain in isolated hepatocytes. *J Pharmacol Exp Ther* 285:480-488, 1978.
4. Iwamoto I, **Eaton DL,** Klaassen CD. Uptake of morphine and nalorphine by isolated rat hepatocytes. *J Pharmacol Exp Ther* 206:181-190, 1978.
5. **Eaton DL,** Klaassen CD. Carrier-mediated transport of the organic cation procaineamide ethobromide by isolated rat liver parenchymal cells. *J Pharmacol Exp Ther* 206:595-606, 1978.
6. Iga T, **Eaton DL,** Klaassen CD. Uptake of unconjugated bilirubin by isolated rat hepatocytes. *Am J Physiol* 236:C9-C14, 1979.
7. **Eaton DL,** Klaassen CD. Effects of microsomal enzyme inducers on carrier-mediated transport systems in isolated rat hepatocytes. *J Pharmacol Exp Ther* 208:381-385, 1979.
8. **Eaton DL,** Klaassen CD. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin, kepone and polybrominated biphenyls on transport systems in isolated rat hepatocytes. *Toxicol Appl Pharmacol* 51:137-144, 1979.
9. **Eaton DL,** Stacey NH, Wong KL, Klaassen CD. Dose-response effects of various metal ions on rat liver metallothionein, glutathione, heme oxygenase and cytochrome P-450. *Toxicol Appl Pharmacol* 55:393-402, 1980.
10. **Eaton DL.** Biliary excretion of 2,4,5-trichlorophenoxyacetic acid in the rat. *Toxicol Lett* 14:175-181, 1982.
11. **Eaton DL,** *Toal BF. Evaluation of the Cd/hemoglobin affinity assay for the rapid determination of metallothionein in biological tissues. *Toxicol Appl Pharmacol* 66:134-142, 1982.
12. **Eaton DL,** *Toal BF. A simplified method of quantitating metallothionein in biological tissues. *J Sci Total Environ* 28:375-384, 1983.
13. *Carpenter LA, **Eaton DL.** The disposition of 2,4-dichlorophenoxyacetic acid (2,4-D) in the rainbow trout, *Salmo gairdneri. Arch Environ Contam Tox* 12:162-173, 1983.
14. *Stinson MD, **Eaton DL.** Concentrations of lead, cadmium, mercury and copper in the crayfish *(Pacificasticus lenisculus)* obtained from a lake receiving urban runoff. *Arch Environ Contam Toxicol* 12:693-700, 1983.
15. Woods JS, Fowler BA, **Eaton DL.** Studies on the mechanisms of thallium-mediated inhibition of hepatic mixed function oxidase activity: Correlation with inhibition of NADPH Cytochrome c (P-450) reductase. *Biochem Pharmacol* 33:571-576, 1984.
16. Woods JS, **Eaton DL,** *Lukens C. Studies on porphyrin metabolism in the kidney: Effects of trace metals and glutathione on renal uroporphyrinogen decarboxylase. *Mol Pharmacol* 26:336-341, 1984.
17. Kalman D, **Eaton DL,** Schumacher RS, Covert D. Biological availability of lead in a paint aerosol. I. Physical and chemical characterization of a lead paint aerosol. *Toxicol Lett* 22:301-306, 1984.

18. **Eaton DL,** Kalman DA, *Garvey D, Morgan M, Omenn GS. Biological availability of lead in a paint aerosol. 2. Absorption, distribution and excretion of intratracheally instilled lead paint particles in the rat. *Toxicol Lett* 22:307-313, 1984.
19. **Eaton DL.** *Short Communications:* Effects of various trace metals on the binding of cadmium to rat hepatic metallothionein determined by the Cd/hemoglobin affinity assay. *Toxicol Appl Pharmacol* 78:158-162, 1985.
20. *Monroe DH, *Holeski CJ, **Eaton DL.** Effects of single dose and repeated dose pretreatment with 2(3)-*tert*-butyl-4-hydroxyanisole (BHA) on the hepatobiliary disposition and covalent binding to DNA of Aflatoxin B_1 in the rat. *Food Chem Toxicol* 24:1273-1281, 1986.
21. **Eaton DL,** *Richards JA. Kinetic evaluation of carrier-mediated transport of ouabain and taurocholic acid in isolated rat hepatocytes. Evidence for independent transport systems. *Biochem Pharmacol* 35:2721-2725, 1986.
22. *Holeski CJ, **Eaton DL,** *Monroe DH, *Bellamy GM. Effects of phenobarbital on the biliary excretion of Aflatoxin P_1-glucuronide and AflatoxinB_1-*S*-glutathione in the rat. *Xenobiotica* 17:139-153, 1987.
23. *Monroe DH, **Eaton DL.** Comparative effects of butylated hydroxyanisole (BHA) on the *in vivo* and *in vitro* biotransformation of Aflatoxin B_1 (AFB) in rat and mouse. *Toxicol Appl Pharmacol* 90:401-409, 1987.
24. Geraci JP, Dunston SG, Jackson KL, Mariano MS, *Holeski C, **Eaton DL.** Bile loss in the acute intestinal radiation syndrome in rats. *Rad Res* 109:47-57, 1987.
25. **Eaton DL**, *Monroe DH, *Bellamy G and Kalman DA. Identification of a novel dihydroxy-metabolite of aflatoxin B_1 formed both *in vivo* and *in vitro* in rats and mice. *Chem Res Toxicol* 1:108-114, 1988.
26. Franzblau A, Rosenstock L and **Eaton DL**. Use of inductively coupled plasma-atomic absorption spectroscopy (ICP-AES) in screening for trace metal exposures in an industrial population. *Environ Res* 46:15-24, 1988.
27. *Monroe DH and **Eaton DL.** Effects of modulation of hepatic glutathione on biotransformation and covalent binding of aflatoxin B_1 to DNA in the mouse. *Toxicol Appl Pharmacol* 94:118-127, 1988.
28. *Ramsdell HS and **Eaton DL**. Modification of aflatoxin B_1 biotransformation *in vitro* and DNA binding *in vivo* by dietary broccoli in rats. *J Toxicol Environ Health* 25:269-275, 1988.
29. **Eaton DL,** *Stapleton PL. Simultaneous determination of cytosolic glutathione *S*-transferase and microsomal epoxide hydrolase activity toward benzo[*a*]pyrene-4,5-oxide by high performance liquid chromatography. *Analytl Biochem* 178:153-158, 1989.
30. *Ramsdell HS, **Eaton DL.** Species susceptibility to aflatoxin B_1 carcinogenesis: Comparative kinetics of biotransformation. *Cancer Res* 50:615-620, 1990.
31. *Ramsdell HS, **Eaton DL.** Mouse liver glutathione S-transferase isoenzyme activity toward aflatoxin B_1-8,9-epoxide and benzo[*a*]pyrene-7,8-dihydrodiol-9,10-epoxide. *Toxicol Appl Pharmacol* 105:216-225, 1990.
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35. *Chen ZY, **Eaton DL.** Differential regulation of cytochrome P450 IIB$_1$/2 by phenobarbital in hepatocellular hyperplastic nodules induced by aflatoxin B_1 or diethynitrosamine plus 2-acetylaminofluorene in male F344 rats. *Toxicol Appl Pharmacol* 111:132-144, 1991.
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41. *Hamel DM, *White C, **Eaton DL**. Determination of γ-glutamylcysteine synthetase and glutathione synthetase activity by HPLC. *Toxicology Methods* 1:273-288, 1992.
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43. *Chen ZY, Farin F, Omiecinski CO, **Eaton DL** Association between growth stimulation by phenobarbital and expression of cytochromes P450 1A1, 1A2, 2B$_1$/2 and 3A1 in hepatic hyperplastic nodules in male F344 rats. *Carcinogenesis* 13:675-682, 1992.
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55. *Borroz KI, *Buetler TM, **Eaton DL.** Modulation of γ-glutamylcysteine synthetase mRNA expression by butylated hydroxyanisole. *Toxicol Appl Pharmacol* 126: 150-155, 1994.
56. *Gallagher EP, Wienkers LC, *Stapleton PL, Kunze KL, **Eaton DL.** Role of CYP4501A2 and CYP4503A4 in the bioactivation of aflatoxin B_1 (AFB1) by human liver microsomes. *Cancer Research* 54: 1-8, 1994.
57. *Chen Z-Y, Liu Y-F, *He C-Y, *White CC, **Eaton DL.** Inhibition of cell proliferation by ciprofibrate in GST - Positive rat hepatic hyperplastic nodules, *Cancer Research* 54: 2622-2629, 1994.
58. Kavanagh TJ, Raghu G, *White CC, Martin GM, Rabinovitch PS, **Eaton DL.** Enhancement of glutathione content in glutatione synthetase-deficient fibroblasts from a patient with 5-oxoprolinuria via metabolic cooperation with normal fibroblasts. *Exp. Cell Res.* 212: 69-76, 1994.
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63. *Heinonen JT, Fisher R, Brendel K, **Eaton DL.** Determination of aflatoxin B_1 metabolism and binding to hepatic macromolecules in human precision liver slices. *Toxicology and Applied Pharmacology,* 136: 1-7, 1996.
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D. ABSTRACTS

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136. *Peck EC, P. L. Stapleton PL,. Grollman AP and **Eaton DL.** Activation of aristolochic acid to mutagenic metabolites by human CYPs 1A1, 1A2 and 3A4. Toxicological Sciences 102(1): #6760, 2008.
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139. Smith WE, Tracy JH, White CC, Bammler TK, Xiaoge H, Xiaohu G, Kavanagh TJ and **Eaton DL.** 2009. Disposition of quantum dots in HepG2 cells and primary human hepatocytes. Society of Toxicology Annual Meeting, Baltimore, MD, March. Poster presentation.
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144. *Smith WE, White CC, Brownell JL, Ashfarinajad Z, Hu X, Gao X, Polyak SJ, **Eaton DL,** and Kavanagh TJ. Differential gene expression changes in two human hepatocyte model systems to

quantum dot exposure. Society of Toxicology Annual Meeting, San Francisco, CA, March. SOT Abstracts 2012, #1267.

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146. **Eaton, DL,** and Tsuji, J. Breast Cancer and the Environment: Interaction of genetics, lifestage and the environment. *Society of Toxicology Annual Meeting, San Francisco, Session Chair*. Abstract # 804, 2012.

147. *Schaupp CM, Bammler TK, Byer RP, Terrance J. Kavanagh TJ and **Eaton DL.** Absence of Upstream Consensus Regulatory Element Sequences for Nrf2 in Human Glutathione S-Transferase Genes. *The Toxicologist*, #2025, 2013.

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149. *Chang S, Voellinger, JL, White C, Kelley EJ, and **Eaton DL.** A Tissue-Engineered Rat/Human Liver Microphysiological System for Drug and Chemical Testing. *The Toxicologist* #708, 2015.

150. *Cook1 TJ, Hoekstra JG, Stewart T, Canales KK, Ho P, Salvador AA, Gonzalez-Cuyar LF, Nelson G, Racette BA, Checkoway H, **Eaton DL** and Zhang J, Astroglial Mortalin Is Decreased in the Striatum of Manganese-Exposed Mine Workers and Enhances Neurotoxicity, *The Toxicologist* #2352, 2016.

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152. Eaton DL . Public Health Consequences of E-cigarettes: A Focus on Special Concerns for Youth and Young Adults. Symposium, Society of Toxicology, The Toxicologist, 168, p 501, #3232, 2019.

8. PATENTS AND OTHER INTELLECTUAL PROPERTY

License for rabbit polyclonal antibody towards human Catechol –O-Methyltransferase, UW TechTransfer, 2001.

Patent application # 11/867,299. Sulforaphane and structural analogs as antagonists of the human Pregnane X Receptor (PXR)**,** 2007**.**

9. FUNDING HISTORY:

A. Active Grants

None

C. Completed Grants, 1985-2019
(DL Eaton, PI – excludes institutional and co-Investigator grants)

NIH 5-P30 ES07033 (Kavanagh, Center Director) 04/01/16 - 03/31/20 0.6 Cal Mo. FTE
PI : Terrance J. Kavanagh (2013-2019)
Title: Center for Exposures, Diseases, Genes & Environment (NIEHS Center Administrative Core)
Role: PI 1995-2013; Associate Director, 2013-2018. The major goal of this NIEHS Center Grant is to provide core support to enhance multidisciplinary collaborations among approximately 75 established investigators in the School of Public Health, School of Pharmacy, and the School of Medicine who are investigating the biochemical and molecular basis for human diseases with an environmental etiology.

EPA-G2013-STAR-L1 (Faustman) 12/1/2014 – 11/30/2018 0.6 Cal Mo FTE
Environmental Protection Agency $6,000,000
Title: Predictive Toxicology Center for Organotypic Cultures and Assessment of AOPs for Engineered Nanomaterials
Role: Co-PI on liver microphysiological systems project. The overall goal of this Center is to develop innovative organotypic culture systems to better evaluate the potential for cellular and organ toxicity following exposure to Engineered Nanomaterials (ENMs) within an adverse outcomes pathway (AOP) model.

NSF DGE-1256082 $ 34,227,534 7/29/2015 – 7/31/2018 no FTE
Title: Graduate Research Fellowship Program (GRFP)
Role: Institutional PI
The purpose of this amendment is to support the NSF graduate Fellows on tenure and on partial tenure at the University of Washington.

NIH UH2/UH3 7/1/2012-6/30/2017 0.6 Cal Mo.FTE:
PI: Jonathan Himmelfarb
Title: Integrated Microphysiological Systems For Drug Efficacy and Toxicity Testing in Human Health And Disease: Microphysiologial Systems – Kidney
Role: co-Investigator on UH3 portion. This grant will develop the Nortis microphysiological system to utilized human kidney cells to study drug toxicity and efficacy.

U19 RFA ES -09-011 10/01/2010-9/30/2015 1.8 Cal Mo FTE
PI: Program Director, Terrance Kavanagh
Title: Linking the physical and chemical characteristics of Qdots to their toxicity.

Role: co-PI (with Bill Parks) PI for Project 1 – in vitro toxicology. This consortium program will identify physical and chemical characteristics of quantum dot (Qdot) nanoparticles associated with toxicity to cells and mice using genetic and epigenetic analyses. The In vitro Studies project will correlate the physical and chemical characteristics of quantum dots with quantitative measures of cytotoxicity in human- and mouse-derived model cell systems.

1 R01 GM079280-01A1 (D. Eaton, PI) 9/01/07 – 8/31/11 1.2 Person Months
NIH/NIGMS/NIEHS/NCI $200,000 ADC
Isothiocyanates as specific antagonists of human SXR (currently on one year no cost extension)

Puget Sound Partners for Global Health 07/01/07 – 06/30/08 0.6 Person Months
PSPGH 2007-55, (D. Eaton, PI) $70,000 ADC
Preventing drug-drug interactions in the treatment of TB in HIV/AIDS patients: an animal model

5R25 ES10738, (Eaton, PI) 09/30/00-08/31/08
NIH/NIEHS $248,415 ADC
Environmental Health Sciences as an Integrative Context for Learning

1 U19 ES11387 (Eaton, PI) 09/30/01 - 08/31/08
NIH/NIEHS $560,000 ADC
The FHCRC/UW Toxicogenomics Consortium.

RO1 ES05780-01-17, David L. Eaton, PI: 08/01/87 - 07/31/04 (to 8/01/05);
NIH/NIEHS $266,142 (ADC); $1,584,405 (TDC)
Species differences in biotransformation of aflatoxin

P42 ES04696 (H. Checkoway, Program Director) 04/01/00 - 03/31/05 .5 % FTE
NIH/NIEHS; Award Amount - Total Program $1,989,426 ADC
Superfund Basic Research Program: Effects-Related Biomarkers of Toxic Exposure
DL Eaton, Deputy Director; (Administrative Core)

P42 ES04696 (sub-project) H. Checkoway, Program Director, and Project PI 04/01/00 - 03/31/05
NIH/NIEHS; Award amount: $156,501 ADC: co-investigator, sub-project); 5 %
Environmental and Biochemical Risk Factors for Parkinson's Disease

R01 ES10750 (H. Checkoway PI). Gene/Environment Interactions in Parkinson's Disease
09/01/00 – 06/30/05: co-Investigator; 5%; NIH / NIEHS.

P42 ES04696 Superfund Basic Research Program, DL Eaton, Program Director (04/01/90 – 09/30/99); ~ $2 million ADC (H Checkoway assumed Program Director responsibilities in 1999; DL Eaton remained active as Deputy Director)

P42 ES04696 Superfund Basic Research Program – Project 1: Glutathione as a biomarker of toxic exposure", DL Eaton, Project PI, 04/01-85 – 09/30/96. (TJ Kavanagh assumed PI responsibility in 1996; DL Eaton continued as co-investigator).

R01 AG17635, DL Eaton, PI 0 6/15/99-06/30/04
NIH/NIA $188,383 ADC; $731,755 TDC
Significance of Genetic Variation in Estrogen Metabolism".

R25 ES08030 DL Eaton, PI 08/01/96-07/30/00
NIH/NIEHS $100,000 ADC
Project Greenskate Teacher Training and Dissemination

R01 ES03933 DL Eaton, PI 05/01/86-04/30/99
NIH/NIEHS $150,000 - $200,000 , ADC
Modification of Aflatoxin Disposition by Enzyme Inducers / Effect of Enzyme Inducers on Liver Preneoplastic Lesions,

R25 ES06938, DL Eaton, PI 07/01/94-06/30/97
NIH/NIEHS $100,000 ADC.
Risky Business: Living in a Chemical World

10. CONFERENCES AND SYMPOSIUMS / INVITED PRESENTATIONS

Symposium Speaker, Society of Toxicology 58th Annual Meeting, "This Is Your Teen Brain on Drugs: In Search of Biomarkers Unique to Dependence Toxicity in Adolescents". Topic: Public Health Consequences of e-Cigarettes, San Antonio, TX, March 18, 2019.

Seminar Speaker, SUNY-Stony Brook School of Medicine, "Using Microphysiological Systems to Identify 'Organ-Organ Interactions' in Toxicology: Effect of the Liver on Aristolochic acid Nephrotoxicity as a proof of concept", June 25, 2016.

Invited Speaker, NIEHS-SOT 50th Anniversary Celebration, NIEHS, RTP, NC, July 13, 2016.

Kopriva Science Lecture, What Scientists Know- and Don't Know-About the Causes of Cancer; Montana State University, Bozeman, MT, Oct. 8, 2013.

Keynote Speaker, Health Effects Institute Annual Meeting, Boston, MA, Gene-Environment Interactions in Air Pollution Research: Challenges and Opportunities, May 2, 2011.

Kuna Distinguished Annual Lecture, Environmental and Occupational Health Sciences Institute, Rutger's University/Robert Wood Johnson Medical School, Piscataway, NJ, May 6, 2010.

Invited Speaker, 10th Annual John Doull Symposium, University of Kansas Medical Center, *Modulation of Aflatoxin-DNA binding by phytochemicals in human hepatocytes* Sept. 3, 2009

Invited Speaker, NAS/IOM Workshop, *Environmental Health Science Decision Making: Risk Management, Evidence, and Ethics*, National Academy of Sciences, Washington DC. January 15, 2008,

Invited Representative (UW), Science and Technology in Society (STS) *forum*: "Harmony with Nature" and "Innovation", Kyoto, Japan, Oc.t 7-9, 2007.

Invited Speaker, *The NAS and WHO on Dioxin and Dioxin Like Compounds: International Policy Implications and Potential Impact*, Michigan State University / NIEHS Superfund Basic Research Program Conference, Sept. 19, 2007.

Invited Participant, Western Library Association Annual Conference, 'Open Access', Tucson, AZ, Sept. 16-17, 2007.

Invited Speaker, NAS/NRC Workshop, *Quantitative Approaches to Characterizing Uncertainty in Human Cancer Risk Assessment Based on Bioassay Results* National Academy of Sciences, Washington DC, ,June 5, 2007.

Invited Speaker, Glutathione S-Transferases, Drug Metabolism short course, American Association of Pharmaceutical Scientists Annual Meeting, San Antonio, TX, Oct. 29, 2006.

Invited Speaker, Presidential Symposium, AAS Annual Meeting: "Risk-Risk Trade-offs: Public Health Examples- 'Contaminated' Drinking Water", ST. Louis, MO, February 18, 2006.

Seminar Speaker, University of Wisconsin-Madison, Interdisciplinary Toxicology program, Oct. 20, 2005.

Discussion Leader, National Institute of Environmental Health Sciences Strategic Planning meeting, Oct. 18-19, 2005.

Invited Speaker, "Functional Genomics and Public Health Protection in the 21st Century", 25th Anniversary Celebration of the National Toxicology Program, National Academy of Sciences, Washington, DC, May 11, 2005.

Organizer and Moderator, Issues Session, Society of Toxicology annual meeting; Reorganization of the NIH Grant review process and its potential impact on toxicology research and training. New Orleans, LA, March 9, 2005.

Invited Speaker, Agricultural Health Study Biomarker Workshop on Cancer Etiology, RTP, NC, sponsored by National Cancer Institute; March 2-3, 2005.

Invited Participant, Workshop on Genetics and Environmental Regulation, Arizona State University College of Law, Tempe, AZ, Jan. 13-14, 2005.

Invited Speaker, NAS/IOM Conference on Implications of Genomics for Public Health, "Gene x Environment Interactions", Oct. 7, 2004, National Academy of Sciences, Washington, DC.

Invited Speaker, North American Congress of Clinical Toxicology, "Liver Cancer", Continuing Medical Education seminar on Insights into the Pathogenesis and Treatment of Toxin-induced Liver Disease", Seattle, WA, Sept. 10, 2004.**Session Chair,** SOT/SETAC Pelston Workshop on "Emerging Molecular and Computational Approaches for Cross-Species Extrapolations", held in Portland, OR July 18-22, 2004.

Symposium Chair, Novel approaches to engaging toxicologists in K-12 science education and outreach, Society of Toxicology Annual Meeting, Baltimore, MD, March 25, 2004.

Invited Commentary, NTP Board of Scientific Counselors Working Group on the National Toxicology Program Vision for the 21st Century, Baltimore, MD, March 25, 2004.

Workshop Speaker, Society of Toxicology Annual Meeting, "Working with Congress", Baltimore, MD, March 23, 2004.

Public Speaker, What Scientists Know-and Don't Know About the Causes of Cancer, Barrow, Alaska, Sponsored by Barrow Arctic Science Consortium; July 29, 2003.

Invited Participant, WHO/ICPS Planning Committee for 'Environmental Health Criteria Document on Principles for Evaluating Health Risks in Children Associated with Exposure to Chemicals', Seattle, WA July 24-25, 2003.

Invited Speaker, Science for Judges, "Scientific Judgment and Toxic Torts", March 28, 2003, Brooklyn Law School, Brooklyn, New York.

Co-Organizer, Molecular and Genetic Epidemiology of Cancer, AACR/SOT sponsored meeting, "Impact of Molecular Epidemiology" (Session Chair), Jan 18-22, 2003, Kona, Hawaii.

Invited Speaker, "Toxicology Training in the 21st Century", presented at a NIGMS Workshop on Pharmacological Sciences Training Program Needs, Bethesda, MD, 8/06/02

Seminar Speaker, University of New Mexico, School of Pharmacy, 4/22/02

Seminar Speaker, Columbia University School of Public Health, 4/16/02

Seminar Speaker, New York University, Nelson Institute for Environmental Medicine, 4/15/02

Sitlington Distinguished Lecturer in Toxicology, 2001, Oklahoma State University, "Molecular basis for species and interindividual differences in susceptibility to carcinogens: Aflatoxin B1 as an example", Stillwater, OK 11/08-09/2001.

Welcome and Introduction, "Use of Genomic Information in Risk Assessment", Current Concepts in Toxicology workshop, Bethesda, MD, Nov. 6-7, 2001.

Invited Speaker, American College of Occupational and Environmental Medicine annual meeting, "Use of genomic information in Environmental and Occupational Medicine", Seattle, WA. Nov. 11, 2001

Invited Speaker, National Capitol Chapter, Society of Toxicology, "New Directions in Toxicology Education for the 21st Century", Washington, DC, May 15, 2001.

Invited Speaker, "Understanding the Science of Toxicology", for 'New Directions in Expert Testimony: Scientific, Technical and Other Specialized Knowledge Evidence in Federal and State Courts', American Law Institute-American Bar Association, San Francisco, CA, April 28, 2001.

Seminar Speaker, University of Colorado Health Sciences Center, "Molecular Basis for Species Differences in Aflatoxin Carcinogenesis", Feb. 21-22, 2001.

Invited Speaker, "Environmental Pollution and Disease: Fact and Fiction", Practical Primary Care Conference, Billings, MT, Oct. 27, 2000.

Invited Speaker, "Susceptibility to Disease and Human Genetic Variation: Implications of the Human Genome Project to the Practice of Medicine", Practical Primary Care Conference, Billings, MT, Oct. 28, 2000.

Invited Speaker, Interindividual Differences in Response to Chemoprotection Against Aflatoxin-Induced Hepatocarcinogenesis: Implications of Human Biotransformation Enzyme Polymorphisms", Sixth International Symposium on Biological Reactive Intermediates, Chemical and Biological Mechanisms In Susceptibility to and Prevention of Environmental Diseases, Paris, France, Université René Descartes, July 16-20, 2000.

Invited Speaker, "Genetics of Susceptibility to Environmental Factors", International Conference on Arctic Development, Pollution and Biomarkers of Human Health, Anchorage, AK April 30-May 3, 2000.

Invited Discussant, "Legal Liabilities at the Frontier of Predictive Genetic Testing", 2nd Annual Arizona State University-Smith Kline Beecham Conference on Genetics and the Law, Phoenix, AZ, April 7-8, 2000.

Seminar Speaker, University of Florida, The molecular Basis for species differences in carcinogenicity of the dietary carcinogen, aflatoxin B1, Jan. 21, 2000, Gainsville, FL.

Invited Speaker, 36th Annual Hanford Life Sciences Symposium, "Individual Susceptibility and Genetic Polymorphisms", Oct. 19, 1999, Richland, WA.

Invited Speaker, American Chemical Society Continuing Education Course, Principles of Toxicology, "Quantitation in Toxicology", and "Basic Principles and Approaches to Experimental Animal Testing in Toxicology", April 27, 1999, San Francisco, CA.

Invited Speaker, Society of Toxicology, High School Teachers Program, Paracelsus Goes to School, "Genes, The Environment and Cancer", March, 15, 1999, New Orleans, LA

Invited Speaker, Society of Toxicology Undergraduate Minority Student Program, "How Chemicals Act in the Body, March 14, 1999, New Orleans, LA

Seminar Speaker, University of Utah, " Molecular basis for species differences in aflatoxin carcinogenesis", November 23, 1998, Salt Lake City, UT.

Invited Discussant, "Genomic Research on Populations Exposed to Environmental Toxins: Ethical, Legal and Social Issues", Nov. 13-14, 1998, Boston, MA.

Seminar Speaker, University of Texas-Houston Medical Center, "Molecular basis for species differences in aflatoxin carcinogenesis", October 13, 1998, Houston, TX.

Invited Speaker/Session Chair, International Neurotoxicology Conference, Pesticides and Susceptible Populations, "Biotransformation Enzyme Polymorphisms and Pesticides Susceptibility", Sept. 13, 1998, Little Rock, AR.

Invited Speaker, Toxicology Forum, "Genetic Polymorphism in the human Glutathione S-transferases: Implications for Human Health", July 12-18, 1998, Aspen, CO.

Invited Speaker, American Chemical Society Continuing Education course "Toxicology for Chemists", San Francisco, CA; April 1-3, 1998.

Symposium Chair, Society of Toxicology Annual Meeting, "The Epidemiology of Breast Cancer: Unraveling the Roles of Genetics, Lifestyle and Environmental Factors", March 5, 1998.

Invited Speaker, "Cancer, Genes and the Environment", lecture in high school workshop "Paracelsus goes to school", Society of Toxicology Annual Meeting, March 2, 1998.

Invited Speaker, Society for Quality Assurance 13th annual meeting, "Liaison Organizations: Society of Toxicology", October 23, 1997, Seattle, WA.

Invited Speaker, NATO Advanced Studies Institute, "Molecular and Applied Aspects of Oxidative Drug Metabolizing Enzymes", August 31-Sept. 11, 1997, Antalya, Turkey

Seminar Speaker, Toxicology Training Program, Department of Environmental Medicine, University of Rochester, May 28, 1997

Symposium Co-organizer and Speaker, Society of Toxicology Annual Meeting, "Genetic Polymorphisms in the Glutathione-S-Transferases", March 10, 1997.

Invited Speaker, ILSI Health and Environmental Sciences Institute, 1997 annual meeting: Emerging Issues in Risk Assessment: Gene-Environment Interactions, Miami Beach, FL, Jan. 21, 1997

Invited Participant, NCI Workshop on Diet, Nutrition, Cancer and Genetic Susceptibility, Jan. 22-23, 1997, Washington, DC.

Plenary Speaker, American College of Veterinary Pathologists, Gene-Environment Interactions: Significance of Human Biotransformation Enzyme Polymorphisms, Dec. 6, 1996, Seattle, WA.

Symposium Speaker, ISSX Annual Meeting: Dietary Modulation of Glutathione S-Transferases, Oct. 20-24, 1996, San Diego, CA

Invited Speaker, Gordon Research Conference on Drug Metabolism, "Class alpha glutathione S-transferases: mechanisms and relevance to variations in human cancer risk", July 7-12, 1996, Holderness, NH.

Zeneca European Lecture Tour: "Molecular basis for species differences in susceptibility to aflatoxin carcinogenesis", University of London, London, England, 8/1/96; Univeristy of Dundee, Dundee, Scotland, 8/8/96; University of Newcastle, Newcastle-Upon-Tyne, England, 8/14/96; Medical Research Council/University of Leicester, Leicester, England, 8/16/96; Zeneca International, Macclesfield, England, 8/18/96; University of Basal, Basal, Switzerland, 9/22/96; Lilly Development Center, Brussels, Belgium, 10/2/96; TNO Nutrition and Food Research Institute, Zeist, The Netherlands, 10/6/96.

Invited Speaker, "The role of cytochromes P450 and glutathione S-transferases in species differences in carcinogen metabolism", International Symposium: Evaluation of Butadiene & Isoprene Health Risks, Blaine, WA, June 27-29, 1995

Invited Speaker, "Genetic polymorphisms as susceptibility factors to environmental pollutants", 47th Annual meeting, American Academy of Forensic Sciences, Medical Toxicology Section Workshop, Seattle, WA February 14, 1995

Seminar Speaker, "Molecular basis for species differences in aflatoxin carcinogenicity", Duke Marine Sciences Center, Beufort, NC July 19, 1993

Seminar Speaker, "Molecular basis for species differences in aflatoxin carcinogenicity", National Institutes for Environmental Health Sciences, Research Triangle Park, NC, April 27, 1993

Seminar Speaker, Toxicology Scholars Colloquium, "Molecular basis for species differences in aflatoxin carcinogenicity", University of Connecticut, Center for Biochemical Toxicology, Feb. 26, 1993

Seminar Speaker, Toxicology Scholar Seminar Series, "Molecular basis for species differences in aflatoxin carcinogenicity", Duke University Integrated Toxicology Program, Feb. 22, 1993

Seminar Speaker, "Molecular basis for species differences in aflatoxin carcinogenicity", University of California-Davis, Feb. 8, 1993

Invited Speaker, Rocky Mountain Academy of Occupational and Environmental Medicine Conference, "Risky Business: Clinical Toxicology & Environmental Health, and Relative Risk Assessment", Jan. 8, 1993

Seminar Speaker, Molecular basis for species differences in aflatoxin carcinogenicity, Chemical Industry Institute for Toxicology, Research Triangle Park, NC, June 4, 1992

Invited Speaker, "Risk Assessment in Managing Environmental Exposure Communications and Media Relations", Medical Group Management Association, Occupational Medicine Assembly Conference, Orlando, FL, February 18, 1992

Invited Speaker, "Principles of Toxicology; Metals Toxicology; Carcinogenesis, mutagenesis and Teratogenesis" lectures, as part of *Risk Assessment, Management and the Communication of Drinking water Contamination*, Continuing Education program sponsored by USEPA and NEHA, Portland, OR, June 27, 1991

Invited Alumni Speaker, "The biochemical and molecular basis for species differences in susceptibility to aflatoxin B_1 carcinogenesis", Department of Pharmacology, Toxicology and Therapeutics, University of Kansas Medical Center, Kansas City, KS April 9, 1991

Invited Seminar Speaker, "The biochemical basis for species differences in aflatoxin carcinogenicity", Department of Environmental Health Sciences, Johns Hopkins University School Hygiene and Public Health, Baltimore, MD, February 12, 1990

Invited Seminar Speaker, "The role of biotransformation in species susceptibility to aflatoxin B_1 carcinogenesis", Medical Research Council, Toxicology Division, Carlshalton, England, Sept. 5, 1989

Participant and Presenter, Third International Conference on Glutathione S-Transferases, "Conjugation of aflatoxin B_1-8,9-epoxide by human liver glutathione S-transferases", Edinburgh, Scotland, August 31 - September 3, 1989

Invited Guest, National Public Radio Broadcast, Discussion on Alar in Apples, June 21, 1989

Invited Speaker, "Pesticides in Our Food and Water. Putting the Risks in Perspective", public information seminar sponsored by the National Environmental Health Association Annual Meeting, Seattle, WA June 25, 1989

Invited Speaker, "Approaches to Risk Assessment and Risk Communication for Forest Use Herbicides", presented to Regional Forest Managers and other agency officials, Portland, Oregon, April 16, 1987

Invited Speaker, "Estimating Occupational and Public Health Risks of Pesticides", Northwest Aerial Applicators Association Convention, Pendleton, OR, November 12, 1986

Invited Speaker, "Toxicology of Wood Preservatives: Pentachlorophenol, Creosote and Arsenicals", given for Montana Department of Agriculture as part of wood preservative applicators' certification training program, Kalispell and Helena, MT, September 30 and October 1, 1986

Session Co-Chairman, "Human Health Implications of Contaminated Seafood", International Symposium on Toxic Chemicals and Aquatic Life: Research and Management; provided closing remarks and session summary, Seattle, WA, September 16-18, 1986

Seminar Speaker, "Effects of enzyme induction on hepatobiliary disposition and DNA binding of Aflatoxin B_1 in the rat", presented at Philadelphia College of Pharmacy and Science, Department of Pharmacology and Toxicology, October 12, 1984

11. UNIVERSITY SERVICE

A. Committees

Member, Executive Committee, President's Population Health Initiative, 2016-18
Member, Activity Based Budget Steering Committee, 2016
Member, International Travel Risk Policy Advisory Committee, 2015-18
Member, Graduation and Retention Task Force, 2015-18
Member, CoMOTION (Formerly C4C) Advisory Council, 2015-18
Chair, Industrial Relations Oversight Committee, 2015-18
Chair, Graduate Tuition Policy Committee, 2014-18
Chair, Review Committee, Dean of the School of Law, 2014-15
Member, Faculty Salary Policy Committee, 2013-2016
Chair, UW Board of Environmental Health and Safety, 2013-2017
Member, President's Task Force on Sexual Assault Prevention, 2013-16
Member, President's Advisory Committee on Enterprise Risk Management (PACREM), 2013-2016
Member, Board of Deans and Chancellors, 2013-2018
Member, Office of Research Proposal Review Committee, 2012-2018
Member, Search Committee, Director, Health Sciences Administration, 2011-12
Chair, Search Committee, Director for School of Fisheries and Aquatic Sciences, 2011-12
Member, UW Ocean Observing Initiative (OOI) Regional Scale Node Advisory Group, 2010-12
Member, Animal Facilities Renovation Advisory Committee, 2011-2013
Member, institutional review committee for School of Nursing, 2011-2012
Member, Search Committee, Dean for School of Dentistry, 2011-2012
Member, Activity Based Budgeting Advisory Committee, 2010-2011
Member, Research Roadmap Oversight Group, 2009-2010
Chair, Department of Environmental and Occupational Health Sciences PhD Oversight Committee, 2009-2010
Chair, Reduced Appointment Policy Committee, 2009-2010
Member, Search Committee, Dean, College of the Environment, 2009-2010
Member, Search Committee, Vice Provost for Global Affairs, 2008
Member, Internal Advisory Board, NHGRI 'Genes and Environment' Statistical Coordinating Center (Bruce Weir, PI), 2008-2018
Member, Internal Advisory Board, NHGRI ELSI Center of Excellence, Center for Genomics & Healthcare Equality, 2006-2012 (PI, Wylie Burke)
Chair, Office of Research Proposal Review Committee, 2006-12
Member, External Advisory Board, Berman Environmental Law Clinic, UW School of Law, 2004-08
Member, Selection Committee, Outstanding Graduate Student Mentor Award, 2000-2006
Research Advisory Board, Office of the Provost, 1999-2006
Member, Public Health Genetics Program Executive Committee, 1999-present
Member, Scientific Advisory Board, NIEHS/UW Center for Child Environmental Health Risks Research, 1999-2015
Affiliate Investigator, Regional Primate Research Center, UW, 1995-2010
Core Faculty Member, Environmental Pathology Training Grant, Department of Pathology, University of Washington, 1980-present

Member, Graduate Faculty, University of Washington, 1979-present
Member, UW Human Subjects Policy Board, 2004-2008
Member, UW Tuition and Fees Policy Advisory Committee, 2006-2007
Member, Search Committee, School of Pharmacy Dean, 2007
Member, UW Data Policy Committee, 2006-2007
Member, Search Committee, Associated Vice Provost for Research-Compliance and Operations, 2006
Member, 5-year review of Dean, School of Medicine, UW, 2004-05
Member, Search Committee, Chair of Department of Biostatistics, School of Public Health, 2004-05
Member, Search Committee for Chair of Dept. Comparative Medicine, School of Medicine, UW
Chair, Search Committee, Director of the Public Health Sciences Division Laboratory, Fred Hutchinson Cancer Research Center, 2003.
Member, Search Committee for Proteomics Faculty position, School of Pharmacy, 2003.
Member, Council of Fellows**,** Pacific Northwest National Laboratories – UW (UW/PNNL) Joint Institutes, 2001-2004
Member, Faculty Advisory Committee for Office of Intellectual Property, 2001-2003
Member, Dept. Health Services Research Committee, 2001-2003
Member, Search Committee for Dental School Dean, 2000-02
Member, Dept. Environmental Health Curriculum & Teaching Policy Committee, 2000-2002
Member, *Ad Hoc* Task Force on Human Subjects, Office of the Provost, 2000-01
Editorial Board, *Northwest Science and Technology*, 1999-2000
Member, Provost's Review Committee for Dean of Pharmacy, 2000
Member, Search Committee, Molecular Epidemiology Faculty position, Fred Hutchinson Cancer Research Center, Public Health Sciences Division, 1999; 2001
Chair, Faculty Council, School of Public Health and Community Medicine, 1998-99
Member, Dean Search Committee, School of Public Health and Community Medicine, 1997-98
Curriculum Committee, Public Health Genetics Program, 1998-
Organizing Committee, interdisciplinary program on Public Health Genetics in the Context of Law, Ethics, and Policy 1997
Member, Friday Harbor Laboratory Advisory Board, University of Washington, 1994-1999
Co-Chairman for Exhibits, University of Washington Health Sciences Open House, 1991
Member, Search Committee for new Chairman of Department of Environmental Health, 1990-91
Member, Civil Engineering Program Review Committee, University Programmatic Review by the Graduate School, 1990
Member, Environmental Studies Review Committee, campus-wide review of Environmental Studies programs and future directions, for the Office of the Provost, 1990
Chairman, University of Washington Technical Oversight and Advisory Committee for Ruston/Vashon Arsenic contamination remedial investigation feasibility study; consultant to Black and Veatch and Washington Department of Ecology, 1987
Member, Search Committee for Occupational Medicine faculty position, 1987
Member, Appointments, Promotions and Tenure Committee, Department of Environmental Health, 1986-1992
Member, Arsenic Pathways Review committee, to assist study group and local health departments interpret and present findings of CDC/UW. Arsenic Pathways Study, 1986
Member, Alcohol and Drug Abuse Institute Executive Council, 1986-1989

Member, Small Grants Review Committee, Alcoholism and Drug Abuse Institute, University of Washington, 1984-86
Chair, Curriculum and Teaching Policy Committee, Institute for Environmental Studies, 1983
Member, Search Committee for Dean of School of Public Health and Community Medicine, University of Washington, 1982
Member, Curriculum and Teaching Policy committee, Department of Environmental Health, 1981-86; Chairman, 1985-86
Member, Search Committee for Director of Laboratories, Department of Environmental Health, 1980.

B. Invited Lectures

UW Law School – *New Student Orientation – 'Perspectives on Expert Witness Testimony', Sept. 18, 2009.*
Dept. Environmental Occupational Health Sciences Seminar, Toxicology and Risk Assessment of Dioxins – the NRC Report, Jan 18, 2007.
School of Public Health Distinguished Faculty Lecturer,, May 25, 2006.
UW Science Forum Speaker (University-wide Public Lecturer): Why me, Doc? What Scientists know – and don't know, about the causes of cancer, April 18, 2006.
Panel Moderator and Discussant, "Research in the Future: UW in 2040", a special symposium in honor of President Lee Huntsman, June 4, 2004.
Invited Speaker, Minority Undergraduate Program in Genome Sciences, Genes, Environment and Cancer, March 9, 2004.
Invited Speaker, Risk Communication: Going 'right to know' to 'right to understand'., "Interpreting and communicating genetic risk information for susceptible populations", April 3, 2003.
Course Director and Speaker, "Environmental Health for Reporters: Ethical and Policy Implications of the Human Genome Project and Genetic Research on Human Sensitivity to Environmental Pollutants", 6/1/2001; "Is it in Your Genes, or the Environment? Why Some People Develop Chronic Diseases and Others Don't", University of Washington.
Program Co-Organizer and Speaker, "Environmental Health For Educators", middle and high school teachers continuing education program, August 1997, August 1998, August 1999.
Invited Speaker, Summer Institute on Ecology & Environmental Sciences, National Science Teachers Association, "Genes, Environment and Cancer", 7/17/96;
Invites Speaker, Science Enhancement for Teachers Program, "Chemicals and Cancer", Biology Teaching Program, UW, May 16, 1996;
Seminar Speaker, "Glutathione S-transferases: Species and individual susceptibility to chemical carcinogens." Department of Medicinal Chemistry, UW, March 11, 1993.
Seminar Speaker, "Delaney Amendment of Carcinogenic Substances". The 1992 Symposium on Science, Technology, and Society, The Influence of Scientific Evidence on National Policy, April 28, 1992
Seminar Speaker, "Glutathione S-transferases: Species and individual susceptibility to chemical carcinogens." Department of Environmental Health, UW, February 5, 1988
Course Co-Organizer and Speaker, "Working With Pesticides: Health and Safety Issues", Northwest Center for Occupational Safety and Health, University of Washington, Seattle, WA, January 16, 1987

Seminar Speaker, "Role of Glutathione-S-Transferase in Detoxification of Aflatoxin B_1", Department of Environmental Health, University of Washington, Seattle, WA, December 4, 1986

Invited Speaker, "Basic Principles of Toxicology", given at "Worker Health and Safety at Hazardous Waste Sites", continuing Education Program, Department of Environmental Health, University of Washington, Seattle, WA, December 4, 1986

Panel Member and Discussant, "Scientist and the Media: Making Headlines on Your Own Terms", February 12, 1986; sponsored by University of Washington Medical Sciences Information Center

Speaker and Program Moderator, Professional exchange on hazardous/toxic substances in and around the home - use and disposal; sponsored by Institute for Environmental Studies, University of Washington, Seattle, WA, June 18, 1984

Invited Speaker, "Chemicals and cancer: Putting the risks in perspective"; Continuing Education Course, Biologists Look at Life and Human Affairs, Biology Program, University of Washington, Seattle, WA, April 16, 1984

Course Organizer and Instructor, "Legal aspects of toxicology"; Continuing Education Course, Educational Resource Center, Department of Environmental Health, University of Washington, SeaTac Hotel, Seattle, WA, December 2-3, 1983

Course Organizer and Instructor, "Principles and practice of toxicology in environmental health"; developed for Continuing Competency Education for Environmental Health Practitioners Program, University of Washington, Seattle, WA, January 25-March 15, 1982

Speaker, "Central nervous system effects of chemicals and physical agents"; Continuing Education Course, Educational Resource Center, University of Washington, Seattle, WA, April 20, 1981

Invited Speaker, "Heavy metals in the environment"; School of Public Health and Community Medicine's 10th Anniversary Celebration, University of Washington, Seattle, WA, September 25, 1980

Invited Speaker, "Occupational toxicology: principles of toxicology"; Department of Environmental Health Continuing Education Program, University of Washington, Seattle, WA, September 19, 1980

Seminar Speaker, "Carrier-mediated transport systems in isolated hepatocytes"; Department of Environmental Pharmacology, University of Washington, Seattle, WA, November 5, 1980

Seminar Speaker, "Effects of trace metals on metallothionein, glutathione, cytochrome P-450 and heme oxygenase activity in rats", Department of Pathology, University of Washington. Seattle, WA, April 11, 1980.

13. PROFESSIONALLY-RELATED COMMUNITY SERVICE

Invited Speaker- Risk Assessment for Dioxins – NRC Panel Experience, US EPA/Wa State DOH, EPA Region 10 Offices, Seattle, Jan. 24, 2007, WA.

Invited Speaker – What Scientists Know- and don't Know- About the Causes of Cancer, Gilda's Club, Seattle, Jan 17, 2007.

Invited Speaker, Seattle Internal Medicine Group, 'Genes, Environment and Cancer", June 17, 2004.

Invited Speaker, "Implications of Genomic Information for Defining Susceptible Human Populations, USEPA, Region X, April 9, 2003.

Invited Speaker, "Risk Communication: going beyond 'right to know' to 'right to understand", talk on Interpreting and Communicating genetic risk information for susceptible populations., Seattle, WA April 3, 2003.

Invited Speaker, Vashion Island Community Meeting, Health Risks from Arsenic in Soil, Vashon Island, WA, Nov. 18, 2002.

Invited Speaker, Washington State Board of Health, Genetics Working Group, "Basic Principles of Genomics in Public Health", Jan. 3, 2002, Olympia, WA.

Course Organizer and Speaker, "Environmental and Occupational Health: A curriculum workshop for middle and high school educators", Northwest Center for Occupational Safety and Health, UW, 7/19/96;

Seminar Speaker, "Small changes in enzyme structure can result in large changes in susceptibility to cancer", Cancer Prevention Research Unit, Fred Hutchinson Cancer Research Center, 7/23/96;

Invited Speaker, Puget Sound Chapter, American Industrial Hygiene Assoc. "Genes, Environment and Cancer: Implications of the Human Genome Project", 3/23/96

Invited Speaker, "Fundamentals of Toxicology", Part 1 & Part 2, 1994 Northwest Federal Safety and Health Conference, Portland, OR, April 27, 1994

Co-organizer and Speaker: "Scientists in the Courtroom: The role of the expert witness", UW School of Public Health and Community Medicine and School of Law, December 9, 1993.

Instructor, Summer Institute in Toxicology, Northwest Center for Public Health Practice, Seattle, WA July 12-13, 1993

Invited Speaker, "Fundamentals of Toxicology", Part 1 & Part 2, 1992 Northwest Federal Safety and Health Conference, Olympia, WA, April 29, 1992

Invited Speaker, "Basic Principles of Risk Assessment", American Public Works Association, Washington State Chapter, Spring Conference, March 26, 1992

Invited Speaker, "Toxic Chemicals, Human Health and the Environment", Science Enhancement for Teachers Program, University of Washington, April 27, 1991

Invited Speaker, "Are rodents good models for human responses to cancer-causing chemicals? Aflatoxin as an example. WEST '91 Conference, Seattle, Convention Center, April 11, 1991

Invited Speaker, "America's Epidemic of Chemicals and Cancer - Myth or Fact?" Inland Empire Agricultural Chemical Association 18th annual convention, Spokane, WA Dec. 11, 1990

Invited Panel Participant, Land Based Marine Pollution in the Pacific Northwest, sponsored by the Ocean Studies Council, University of British Columbia, Vancouver, BC, Nov. 2, 1991

Program Organizer and Speaker, "Human Health Risk Assessment: Process and Limitations," principal lecture for a symposium on uncertainties in risk assessment and risk management, given to upper level managers in the Washington Department of Ecology, March 13, 1990

Invited Speaker, "Risk Assessment in Foods", and Panel participant, "Chemical Residues in Foods", presented at Food Safety in Northwest Supermarkets, sponsored by Washington State University Cooperative Extension, Culinary Botany Northwest, and Department of Environmental Health, UW. October 12, 1989

Invited Guest, "Pesticides in Our Food", The Jim Altoff Show, KING 1090 Radio, October 9, 1989 (1 hour)

Invited Speaker, "Pesticide residues in our food and water- a hazard to your health, or a negligible risk?" Saturday Seminar Series, sponsored by the Office of University Relations and UW Extension, Sept. 30, 1989

Seminar Speaker, "Biochemical basis for Species Differences in Aflatoxin carcinogenesis", Washington State University Pharmacology/Toxicology Program Seminar, July 25, 1989

Invited Participant, "Town Meeting", local television talk show focusing on health controversies over pesticide residues in food, KOMO TV, Seattle, WA, April 13, 1989.

Invited Speaker, "Strengths and Weaknesses of Toxicological Evidence", presented at Continuing Education Program on "Legal Aspects of Occupational Health", University of Washington Education Resource Center, February 8, 1989, Seattle, WA.

Co-organizer and Speaker, "Pesticides in the Urban Environment: Health and Environmental Responsibility", program for 1988 annual convention of the International Pesticide Applicators Association, held in Bellevue, WA on Sept. 28-30, 1988

Invited Speaker, City Club (a downtown business leaders civic group). Discussion of the procedures and difficulties encountered in utilizing science in decision making regarding public health risks. Seattle, WA, February 23, 1988

Invited Speaker, "Utilization of Scientific Information in Risk Management Decisions", presented to Division Mangers and Budget Coordinators for METRO, Battelle Conference Center, Seattle, WA, October 14, 1987

Invited Speaker, "Problems in Risk assessment and Risk Communication", King County Health Department annual retreat, Seattle Center, Seattle, WA, March 18, 1987

Invited Speaker, "Taking Care of Yourself - Pesticide Use and Human Health", presented at 77th Annual Meeting of the Western Washington Horticultural Association, Olympia, WA, January 6, 1987

Invited Speaker, "Toxic Chemicals and Public Health: Putting the Risks in Perspective", presented to the Seattle Chapter of the American Society of Safety Engineers, October 27, 1986

Invited Speaker, "Principles of Toxicology", given at "Toxic Substances and Public Health Workshop", sponsored by the Indian Health Service, Seattle, WA, October 22, 1986

Invited Speaker, "Pesticides and Public Health: Risks and Uncertainties", presented at the International Pesticide Applicators Association Convention, Spokane, WA, October 2, 1986

Invited Speaker, "Basic Principles of Toxicology", Risk Assessment Workshop for State Legislators Olympia, WA. Sponsored by Washington Department of Social and Health Services, September 25, 1986

Invited Speaker, "Basic Principles of Toxicology and Risk Assessment", presented to Community Health nurses and doctors for the Seattle-King County Health Department, Seattle, WA, June 10, 1986

Invited Speaker, "Potential health effects of occupational Exposure to PCB's", presented to Snohomish County PUD utility workers, March 17, 1986

Invited Speaker, "Health Implications of Gaseous Emissions Associated with Midway Landfill", February 6, 1986, Kent, WA; sponsored by Washington State Department of Ecology

Course Co-Organizer and Instructor, "Basic Principles of Toxicology for Environmental/ Health Practitioners"; two day course given in Tacoma, WA (July 16 & 17, 1985), and Spokane, WA (August 29 & 30, 1985)

Speaker, "Toxicology of PBC's, dioxins and dibenzofurans", given at a public meeting in Kitsap County at the request of Seattle City Light Environmental Affairs Division, Seattle, WA, June 17, 1985

Invited Speaker, "An evaluation of the health concerns over dioxins found in Eagle Harbor, WA", given at a community meeting at the request of Dr. Willa Fisher, Director of the Kitsap County Health Department, July 11, 1985

Invited Speaker, "Health Impacts of Environmental Pollutants", given for the Continuing Medical Education Series, Riverton Hospital, July 19, 1985

Invited Speaker, "Health Effects of PCB's", given to Seattle City Light employees on November 1, 1984, January 9, November 19, November 21, December 4, and December 11, 1985

Course Organizer and Speaker, "Toxic Chemicals: Communicating Risks to the Public", forum for Scientist-media exchange, sponsored by the Society of Toxicology and the Department of Environmental Health, Battelle-Seattle Conference Center, Seattle, WA, April 27, 1985

Invited Speaker, Public Perception of Health Impacts of Chronic Pesticide Exposure; International Pesticide Applicators Association Convention, Fife, WA, September 27, 1984

Course Organizer and Instructor, Toxicology Review Course for EPA Personnel; Seattle, WA, September 5, 1984-January 15, 1985 (attended by 45 EPA staff)

Invited Speaker, "Pesticides use for Gypsy Moth control", presented at Controversies in Poisoning Management and Occupational/Environmental Exposures, Children's Hospital and Medical Center, Seattle, WA, April 28-29, 1983

Lecturer, "Recent Developments in Biological Monitoring"; Continuing Medical Education Course, Seattle, WA, March 18-19, 1983

Lecturer, Toxicology; short course presented to Hazards Assessment Program employees of NOAA, 3 lectures; NOAA Sand Point Facilities, Seattle, WA, March 10, 1983

Speaker, Health implications of Gypsy Moth control programs; Public Information Seminar, sponsored by Department of Environmental Health and Institute for Environmental Studies, University of Washington, March 10, 1983

Invited Speaker, "Health effects of chronic pesticide exposure: a training program of health personnel: prevention, recognition and treatment of pesticide-related illness"; sponsored by Yakima Valley Farm Workers Clinic and the Migrant Health Clinics in Washington, Oregon and Idaho, Pasco, WA, January 21-22, 1983

Invited Speaker, "Clinical toxicology of pesticides"; Northwest Poison Control Center Conference, Spokane, WA, April 22-23, 1982

Invited Speaker, "A survey of toxic torts"; Washington State Trial Lawyers Association Annual Meeting, Vancouver, BC, Canada, July 10, 1981

Invited Speaker, "Carcinogens in the environment"; Washington State Office of Environmental Education in Health Education, Everett, WA, March 10, 1981

Invited Speaker, "Phenoxy acid herbicides--fact and fiction"; Huxley College of Environmental Studies, Western Washington University. Bellingham, WA, January 27, 1981

Invited Speaker, "Central nervous system effects of industrial chemicals and physical agents"; 1980 Occupational Health and Medical Conference, Spokane, WA, October 22-24, 1980

Invited Speaker, "PCB's and human health". Washington State Office of Environmental Education and Health Education. Seattle, WA, February 27, 1980.

15. TEACHING HISTORY

A. Teaching (2019-present)

As adjunct Professor of Pharmacology and Toxicology, University of Arizona, R. Ken Coit College of Pharmacy, I teach 2 classes each year in the advanced toxicology lecture series; 1) Adverse Outcomes Pathways Approach to Chemical Risk Assessment, and 2) Mode of Action of Natural Toxins.

B. Teaching (2011-2018):

Updated 03-05-2024

While serving as Dean of the Graduate School, I co-taught, with Dr. Thummel (Chair, Dept. Pharmaceutics) PHG/ ENVH/PCEUT 513, Fundamentals of Pharmacogenetics and Toxicogenomics. Enrollment is typically 20-25 graduate students. I had 50% responsibility for this course.

I also provided 5 lectures each year to the three-quarter graduate Toxicology series, ENV514 (1), ENVH 515 (3) and ENVH 516 (1).

C. Previous Teaching:

In 2012 I adapted a previous ENVH 590A graduate toxicology course to a new format. This was a 4 credit course, with enrollment of 15-20 non-toxicology graduate students. The course utilized the lecture content of ENVH 405, of which I give 50% of the lectures, plus included a 1 hr per week discussion group with the graduate students. I was responsible for assigning the readings for each weekly session, and helping the students who lead the discussion. I pass this course on to others when I became Dean of the Graduate School.

From 1979 - ~1985, I developed and taught a 1 quarter in depth Toxicology course, ENVH 515, with enrollment of ~10- 15 students. I also co-taught ENVS 101, Introduction to Environmental Studies (5 credits 200-300 students) at least once a year from 1979-1983.

From ~1986-1995 I had primary responsibility for teaching one of the three quarter toxicology courses.

From ~ 1990 – 2000 (when I devoted 40% effort as Associate Dean for Research in the School of Public Health, and then the 40% Position as Associate Vice Provost for Research), I had 100% responsibility for ENVH 405, Basic Principles of Toxicology, with typically 30-50 students enrolled. I also had either 50% or 100% responsibility for ENVH 567 – Environmental Carcinogenesis (3 credits, typical enrollment of 6-12 students).

I Taught one Lecture to Public Health Genetics 200 in fall and spring quarters, and undergraduate course with about 80 students for 5 years. The course was led by Dr. Patricia Kuzsler in the Law School.

In 2010 I developed, with Dan Luchtel, a graduate level course in toxicology for non-toxicology EHS doctoral students in occupational medicine and exposure sciences (and other programs). I was 50% responsible for this in 2010 and 2011. The course was restricted in 2012, as discussed above, and I still maintain 50% responsibility for it.

C. Graduate Student Mentoring:

MS/MSPH Students (Preceptor)

Brian Toal	Joel Rank	Misha Trusty
Leslie Carpenter	Denise Hamel	Julie Hill
Margaret Stinson	Karyn Micheleson	Jason VanLoo
Heidi Hagelstein	Ingrid Borroz	Marc Stifleman

Julia Richards	Dana Stahl	Kate Bradley

PhD. Students Mentored

Trainee Name	Training Period	Degree, Date, Institution	Project Title	Current Position
Carolyn Holeski	83-86	BS, UC-Davis, Toxicology; PhD, UW Pathology, 1986	Effects of enzyme inducers on the hepatobiliary disposition of Aflatoxin B1 in the rat	MD in private practice
David Monroe	84-87	UW, Ph.D., 12/87	Species and Diet Related Resistance to Chemical Carcinogens: Aflatoxin B1	Staff Scientist, USEPA, Region VI, Kansas City, MO
Van Ness, Kirk	92-95	MS, 1986, University of Washington	Identification of amino acids in glutathione S-transferase mYc responsible for high activity toward aflatoxin oxide	Research Scientist Iuvo BioScience, Rush, NY 14543
Wang, Charles (Changhong)	94-99	MSPH, 1988; MD, Tongji Medical School	Complementary DNA cloning and sequence of alpha class GSTs in monkey liver	Professor and Founding Director, Center for Genomics, Loma Linda University School of Medicine, Loma linda, CA.
McHugh, Tom	94-98	MS, , 1993, Stanford University	Molecular characterization of glutathione transferase with high activity toward aflatoxin 8,9epoxide	Principal Toxicologist, Groundwater Services International, Houston, TX
Smith, Helen	98-2004	BS, U. Texas-Austin, Pharmacy, 1989; MS, Community Health, UT-Houston, 1994; Ph.D. UW, 2004	Polymorphisms in estradiol metabolism in human endometrium	Professor, School of Pharmacy, University of the Incarnate Word, San Antonio, TX
Abel, Erica	99-2004	BS, Texas A & M, 1997; Ph.D. UW, 2004	Structure-function studies on glutathione S-transferases	Clinical Professor of Biology, Baylor University, Waco, TX

Guo, Yingying	99-2004	MD, Sun Yat-sen University, Gungzhou Province, China, 1994; MS, Univ. Cincinnati, 1998; Ph.D. UW, 2004	Effects of aflatoxin epoxide mediated DNA damage on global gene expression in yeast and HepG2 cells and human hepatocytes.	Research Scientist, Eli Lilly & Co, Indianapolis, IN
Poulton, Emma-Jane	2004-2010	B.S., Chemistry, 2004, MIT	The phytochemical, sulforaphane is a sensitive and specific inhibitor of the human steroid X-receptor (SXR)	Senior Scientist, Sanofi Pharmaceuticals, Framingham, MA
Peck, Erin	204-2010	B.S., Chemistry 2002, Univ. North Carolina, Chapel Hill	Role of human CYP1A2 in the activation and detoxification of aristolochic acid	Primary Care Physician, Duke Medical Center, Durham, NC
Vandivort, Tyler (co-mentor with Bill Parks, Pulmonary Medicine)	2011-15		The Function and Regulation of Macrophage Matrix Metalloproteinase 10 (MMP10) in Lung Injury and Fibrosis	Director, Regulatory Affairs and Operations, Amplicore Inc., Cincinnati, OH.
Cook, Travis (co-mentor with Jing Zhang, Pathology)	2011-2014		Mechanisms of Mn-induced Parkinson's disease	Lead Toxicologist, Product Safety, SABIC Corp., Houston, TX
Shi-Yu (Shirley) Chang	2012-2016		Development of a microphysiological system using human liver cells	Toxicologist, Janssen Pharmaceuticals Company of Johnson & Johnson

Post-doctoral Fellows Mentored:

Trainee Name	Training Period	Previous Degree, Date, Institution	Project Title	Current Position
Ramsdell, Howard	87-89	Ph.D., 1989, Oregon State University	Biochemical basis for species differences in aflatoxin biotransformation	Assoc. Professor, Dept. Environ. Hlth, Colo. State Univ., Fort Collins, CO
Buetler, Timo	90-94	Ph.D., 1989, University of Basel, Switzerland	Molecular cloning and biochemical characterization of alpha class glutathione transferase in mouse liver	Senior Scientist, Nestle, Inc., Basal, Switzerland
Chen, Zhi-Ying	90-95	1970, M.D., Sun Yat Sen University	Effects of enzyme inducers on liver preneoplastic lesions	Research Scientist, UCLA School of Medicine
Gallagher, Evan	91-94	Ph.D., 1991, Duke	Kinetics of CYP1A2 and 3A4-mediated aflatoxin activation and detoxification	Professor Emeritus, Environ. Occup. Health Sciences, UW, Seattle, WA

Bammler, Theo	95-2000	Ph.D., University of Dundee, Scotland	Effects of Oltipraz and ethoxyquin on disposition of aflatoxin B1 in the marmoset monkey	Res. Scientist, Manager of Microarray Facility, Center for Ecogenetics, UW, Seattle, WA
Kelly, Edward	96-2001	Ph.D., 1996, Univ. Washington	Role of human epoxide hydrolase in the detoxification of aflatoxin B1	Assoc. Professor, Dept. Pharmaceutics, UW, Seattle, WA
Gross-Steinmeyer, Kerstin	2002 - 06	PhD	Phytochemical perturbations of aflatoxin biotransformation	Currently 'at home' mother
Smith, Wesley	2008-2012	PhD, Univ. Montana	Toxicology of QDot nanomaterials	Risk Assessor, California State OEHHA